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OBSERVATIONS OF SURGICAL TRAINING IN AMERICA IN RELATION TO THE OBJECTIVES OF THE AMERICAN BOARD OF SURGERY

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THE attention of physicians contemplating a career in surgery is directed almost automatically toward the American Board of Surgery and its requirements for examination. Although the Board has never been concerned that those receiving certificates be granted special privileges, the desire by trainees for certification has become more intense each year since the Board's organization in 1937. This zeal for certification has at times operated to the detriment of the trainee, as the candidate has concerned himself almost exclusively with the mere acquisition of the minimum requirements for examination. At that point, despite the opportunities which might present themselves, training has all too frequently terminated. Proof of this attitude is reflected in the volumes of correspondence in the Board office which include the questions, "How many months must I remain here?" "Will three months do it, or will it take six?"

Prior to the establishment of the Board, the opportunities for satisfactory graduate surgical education were limited, despite the fact that Halsted had introduced the resident system in America at about the turn of the century. At first the Board examined appropriate candidates and issued certificates to those who passed its objective examination. The educational requirements for examination were considerably less formal than those at present in effect. In the period 1937 to 1946, many candidates became eligible for examination under the preceptorship plan. During this period the examinees for the most part had completed their surgical education and many were already established in the practice of surgery. It would have been unreasonable during that period to have demanded formal residency training and, in so doing, eliminate from examination and certification the great bulk of practicing surgeons.

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During the period from 1937 to 1946, eligibility for examination could be realized without residency training provided the candidate had completed five years of acceptable training in surgery. Although many excellent surgeons were developed under that system, it soon became apparent that good residency training in suitable institutions offered the greatest number the best opportunity to attain surgical competence. All too frequently the arbitrary relationship established between preceptor and preceptee was less than ideal. Patient and operative responsibility developed slowly or not at all. At times the threat of impending professional competition was eliminated by a dissolution of the association. An analysis of the candidates and their performance at examination convinced the Board that those men who had had the advantages of good residency training were, in general, much better prepared. The validity of this observation has been substantiated by the rapid development of the residency system during the last ten years.

In 1947 the educational requirements were altered so that residency training became essential in meeting the requirements of the Board. A complete description of the present educational requirements for examination is available in booklet form and will not be discussed here. In brief, however, all candidates at present are expected to complete not less than three years of residency or fellowship training in surgery in approved hospitals. The additional time required over and above this may be spent in surgical research, basic science, graduate schools of medicine, or in supervised practice of surgery. During the period of formal training, it is hoped that the resident will assume ever-increasing positions of responsibility. The Board recognizes the danger of over-regimentation in the educational program and for this reason has made a real effort to include elasticity consistent with good training in its educational requirements. Almost all surgeons are agreed that three years of clinical surgery taken either as a resident or a fellow represents the minimum time required to develop a surgeon who will be expected to display good judgment before, during, and after operation. Complete freedom is extended to the candidate with respect to training in basic science. This arrangement is certainly not too rigid. In a word the Board demands knowledge in the basic sciences related to surgery, but does not concern itself with the manner by which such information is obtained. This liberal attitude with respect to the basic science requirement is not intended to detract from their fundamental importance. It arose from the desire to give both candidate and surgical director a freedom of action so that more attention might be devoted to training than to the development of a schedule. It may be argued that if such a liberal attitude is reasonable with respect to the basic sciences, it might apply with equal effectiveness to clinical surgery. Such an argument carried to the ultimate would eliminate all requirements and thereby place unwarranted dependence upon examination.

It is sometimes stated that the progress of the better-than-average student is stultified by Board requirements. He feels that if he is to be certified at a reasonable age that he cannot afford to spend time in any worth-while pursuit

unless he is assured of Board credit. Since the Board insists only upon minimum requirements, there is serious doubt whether this demand ever adversely affects a surgical career. Experience has shown that most of the above-average men realized Board requirements in the pursuit of their studies, even if they are totally unfamiliar with the requirements. There can be little doubt that the average and below-average student, as a result of these requirements, has seen fit to obtain at least minimum training and has as a consequence become more proficient in the field. If the requirements are not too rigid, the candidate is oriented in the general direction of surgical competence. The paths which he may follow are many, but all point in the same general direction. Without any guidance, many would emerge into surgical practice with little or no training. It is believed that the trial-and-error methods in the acquisition of surgical skill exact from society a needlessly high price.

There are those who sincerely feel that educational methods are not within the province of the specialty boards. If such be true, on whom should this responsibility now fall? The function of the undergraduate medical school is the development of broadly trained doctors of medicine, with special emphasis on medicine, surgery, obstetrics, and pediatrics. It is not concerned with the training of specialists. Postgraduate schools of medicine at present offer only segments of specialty training. If in the future more complete programs of special training are provided by these schools, the Board will take an increasingly liberal attitude in this matter.

The insistence upon residency or fellowship training as an absolute essential in the realization of Board requirements imposes a moral obligation upon the Board to make certain that institutions which have approval for such training provide at least satisfactory training. The Board has a responsibility to the candidate, who in good faith embarks upon a three- or four-year program in surgery, to make reasonably certain that such institutions will offer acceptable training. This responsibility exists whatever body or combination of bodies concern themselves with hospital standardization.

There are those who feel that the Board should content itself exclusively with the conduct of examination and the issuance of certificates. The objections to this thesis are several. One of the purposes of the Board is the establishment of principles of surgical education made applicable to young men who contemplate a surgical career. This purpose would be nullified if the Board concerned itself exclusively with the conduct of the examination. The acceptance of this thesis places greater faith on the reliability of examination than deserved. All educators are agreed that, however perfect an examination, it will leave much to be desired. Surgical judgment, certainly a most important attribute of a surgeon, lends itself poorly to assessment by examination alone. There can be little doubt that many candidates, surgically incompetent, could with concentrated effort prepare themselves successfully for a given examination. For these reasons, the Board has seen fit to establish and exact the minimum training requirements and places as much emphasis on training as on the ability to pass the examinations.

The desire for surgical residencies has increased greatly and it must be apparent that the American Board of Surgery has played some role in this demand. The number of deserving young men who desire residency training in surgery greatly exceeds the capacity of institutions prepared to offer such training. This simply means that hospitals which receive approval experience no difficulty in filling their vacancies. The activity initiated when a hospital is dropped from the approved list, out of all proportion to that exhibited in years past, need not, and does not, necessarily signify a sincere desire on the part of the hospital and its staff to improve the quality of training. For this reason, appropriate bodies responsible for accrediting residencies in hospitals have in the past and will continue in the future to include only those hospitals on the approved list which offer at least satisfactory training in surgery. That errors have been made in the past, both positively and negatively, cannot be denied, but it is anticipated that with time these injustices will be corrected.

Under the resident system, the candidate has never expected more than a stipend for his services. This has been acceptable on the basis of professional service for surgical training. When good surgical training is rightfully anticipated but not provided, the whole process may be labeled "exploitation." The candidate accumulates paper credit in a paper program and frequently emerges with a limited knowledge in the field of surgery. The desire for residents is widespread—the desire to train them somewhat less.

A good training program, however excellent its physical facilities and ancillary services, cannot function unless adequate clinical material and qualified teachers with time and the desire to teach are provided. One of the essential considerations in the operation of a training program entails a careful selection of applicants for appointment to the resident staff. This responsibility usually rests upon the department head. It would seem, however, that in the selection of applicants perhaps undue emphasis has been placed on previous scholastic standing. There can be little objection to the method, provided, of course, that at least some attention is directed to those other qualities embraced in the word "character." Honesty, perseverance, loyalty, dependability, and common sense are certainly attributes which merit some consideration.

The resident system was introduced to provide the surgeon in training with ever-increasing patient and operative responsibility so that in the final year major responsibility might be borne. This type of training cannot be obtained in institutions devoted exclusively to the care of private patients, although there are those who feel that training may be pursued just as effectively by observation of and assistance to a capable surgeon. All are agreed, however, that this responsibility must eventually be assumed, and it seems reasonable that if this is gradual and progressive during the early formative years, it would be advantageous. The bulk of the operative work on ward patients in a hospital organized for graduate training in surgery that maintains residents for three or four years should be performed by the resident staff. If the resident in his final year is unequal to the task, it must signify improper selection of

applicants, or poor training, or both. If on the other hand, the resident staff is competent, there is little reason to deny them this privilege. There are certain patients who may deserve the attention of the more experienced surgeon, but in the average institution this does not comprise more than about 15 per cent, if that much. The excessive diversion of the operative work from the resident to the junior staff may spring from the fact that the department head, cognizant of the weakness of his junior staff, has embarked upon a program of staff education. Such a system is designed primarily for staff training, not residency training, and should be so identified.

A great deal of discussion and some serious thought has been given to the continuing segmentation of surgery into an ever-increasing number of subspecialties. While I do not wish to consider the problem at length, I believe there can be little doubt that if this trend continues indefinitely, it will operate to the detriment of the developing surgeon. The over-subdepartmentalization in surgery is likely to reach its greatest height in the university hospital. It is not uncommon to find surgeons emerging from a four-year surgical program totally unacquainted with the problems of head trauma. While cranial trauma lies squarely within the province of the neurosurgeon, the services of a neurosurgeon are not readily available in many sections of the country. The broadly trained general surgeon should be prepared to handle the problem at least temporarily. Other comparable examples could be cited indefinitely. In an effort to acquaint the resident in general surgery with subspecialty problems, the rotation of residents through the subspecialties has sprung into practice. As the number of subspecialties has increased, this rotation has become more troublesome, if training is not to continue indefinitely. Until there is a better distribution of specialists with particular fields of interest, many of these problems will necessarily come to the attention of the general surgeon. While there can be little doubt, other factors remaining equal, that the limitation of one's interest to a given anatomic region or system may result in greater efficiency in that field, there is grave danger that one's attention may be confined too early and too completely. As a consequence, surgical growth is stunted. From the standpoint of the patient's welfare there must be a point where one may know too much about too little.

Surgical principles are principles which apply to all branches of surgery. The problems of wound-healing, infection, shock, tissue reaction to foreign material, are not peculiar to any one surgical field. The specialty boundaries are, at best, poorly demarcated and extend imperceptibly into adjacent fields of interest. This overlapping is inevitable so long as the patient remains intact. Disease progresses without thought of the specialist. The neoplastic fusion of the reproductive organs with portions of the alimentary tract require the services of a broadly trained surgeon appreciative of and equal to the problems of both systems, whether he be gynecologist or general surgeon. The transplantation of ureters into the bowel poses no special problem to the surgeon familiar with the technical, anatomic, pathologic, and physiologic features of

both systems, whether he be urologist or general surgeon. The heated discussions as to "who does what" are of minor concern to society so long as surgeons recognize and respect their limitations.

Whether or not the specialty boards have exerted a favorable influence on medical and surgical practice will be answered more accurately in the succeeding ten years. Specialty board development has been attended by undesirable side-effects and it is the responsibility of the boards to recognize and eliminate these detrimental influences as completely as possible. At present, it would appear that the good effects of the specialty boards on American medicine outweigh the evil. Unwarranted stress has been placed upon certification in the assignment of personnel to staff and teaching positions. It is not the problem of the Board to specify requirements for staff membership. The ruthless dismissal of accomplished surgeons, who happened not to have been certified, from positions of responsibility, is not justified. The Board does not attempt to decide who shall or shall not operate. Surgical competence does not spring automatically from certification. There are many capable surgeons whose excellence springs neither from certification nor from the absence of it.

THE SEARCH FOR LYMPH NODE METASTASES IN CANCER OF THE RECTUM

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INTRODUCTION

DURING the past 20 years, the behavior of cancer of the colon as it grows and metastasizes has been made the subject of intensive study.[1-14] The technique for preparing the specimens for an examination of the lymph nodes has generally been a modification of the methods used by Spalteholz[16] for preparing translucent anatomic preparations. The value of such methods is well attested by their wide adoption and by the general agreement of data obtained by various investigators.

When the program of secondary delayed re-entry of the abdomen in patients exhibiting lymph node involvement was instituted at University Hospitals,[17] the need for a more complete examination of the specimens removed at the initial operation became evident. In an effort to fill this need, the method of Spalteholz as modified by Gilchrist and David[10] for the examination of the lymph nodes in patients with cancer of the colon was investigated in order to determine its usefulness and practicality in our own laboratory. The examination of 20 specimens of cancer of the rectum removed by combined abdominoperineal excision forms the basis for this report.

METHOD

The fresh bowel specimen was perfused with a solution of potassium citrate (1 per cent) through the superior hemorrhoidal artery to remove as much blood as possible. It was then opened longitudinally, fastened on a wire frame, and fixed in a formalin solution (10 per cent). After 48 hours the specimen was placed in a solution of alcohol (50 per cent). The concentration of the alcohol was slowly increased until the specimen was thoroughly dehydrated in absolute alcohol. It was then placed in methyl salicylate and allowed to clear. This entire procedure required about 2 weeks.

When clear, the specimen was carefuly dissected, transmitted light being used, and all of the lymph nodes were removed. The nodes were measured and placed in individually labeled vials, and their position was recorded upon a diagram. The nodes were then blocked in paraffin, sectioned, and stained with hematoxylin and eosin. An average of 5 sections from each node was prepared for examination. After microscopic examination, the presence or absence of tumor cells was recorded. From these data, the topography of the specimen was constructed.

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RESULTS

The number of nodes examined, the number of nodes found to contain tumor cells, and the Dukes' classification[5] of the 20 cases are shown in Table I. A total of 1,048 nodes, an average of 52.4 in each specimen, was examined, and 122 were found to contain tumor cells. The position of the positive lymph nodes in relation to the primary tumor was quite variable, in some cases being contiguous and in others well up the mesentery and without involvement of the intervening nodes (Fig. 1).

The relation of the size of the lymph node, measured across its largest diameter, to the presence of tumor cells is shown in Table II. No metastases were

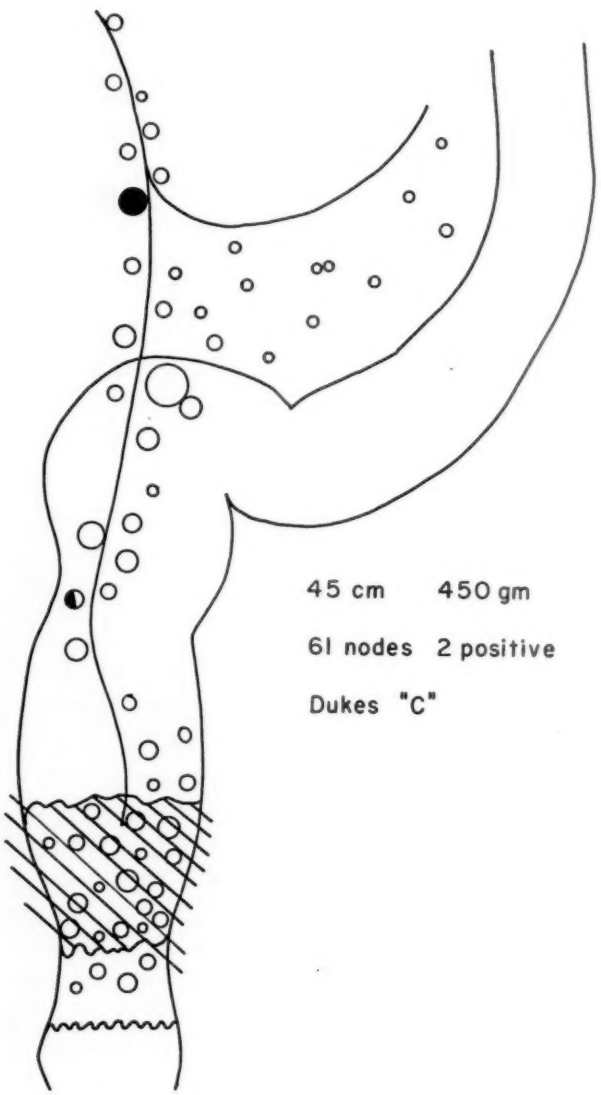

Fig. 1.—This figure demonstrates the topographic reconstruction of specimen No. 6 and demonstrates the position of the lymph nodes containing tumor cells. The solid black node denotes established tumor growth; the half-blackened node contains a tumor embolus.

found in lymph nodes less than 2 mm. in diameter. These nodes made up more than one-third of the total number examined. Eleven nodes of the 122 containing tumor cells measured 3 mm. in diameter. In each case in which such a small lymph node was found there were tumor cells; in others, larger nodes were found to be involved also. In other words, not one of the cases with lymph node metastases would have been overlooked if nodes 3 mm. in diameter and smaller had been discarded. Over one-half (638) of the total nodes examined (1,048) were 3 mm. in diameter or smaller.

TABLE I. A TABULATION OF THE NUMBER OF NODES EXAMINED, THE NUMBER FOUND TO CONTAIN TUMOR CELLS, AND THE DUKES' CLASSIFICATION IN 20 CASES OF CANCER OF THE RECTUM

CASE NUMBER	NUMBER OF NODES EXAMINED	NUMBER OF POSITIVE NODES	DUKES' CLASSIFICATION[5]
1	20	0	A
2	84	35	C*
3	17	4	C
4	53	2	C
5	70	0	A
6	61	2	C
7	39	0	B
8	43	1	C
9	55	5	C*
10	53	0	B
11	47	0	B
12	48	0	B
13	15	4	C
14	68	15	C
15	55	1	C
16	23	10	C
17	51	0	B
18	78	28	C
19	98	5	C
20	70	10	C
Totals	1,048	122	

*Patients having liver metastases at operation.

DISCUSSION

The data accumulated in this study of cancer of the rectum conform completely with the data of Gilchrist and David[3, 9-12] in regard to the average number of lymph nodes found in each specimen, the number and topography of the nodes found to be involved with tumor, and the histologic pattern of the cancerous involvement in the lymph node.

No information is available in the literature concerning the relationship between the size of the node and the presence or absence of metastases, although it appears to be of considerable importance. This is especially true if it can be established that metastases rarely, if ever, occur in lymph nodes smaller than 2 mm. in diameter.

According to the best estimate, tumor emboli are relatively large (100 to 200 μ).[18] How many sections (15 μ) should be made from each lymph node in order that no such emboli be overlooked? Saphir and Amromin[15] made serial sections and looked at every other section. What is the probability of missing

TABLE II. THE PRESENCE OR ABSENCE OF LYMPH NODE METASTASES IN RELATION TO THE SIZE OF THE LYMPH NODE

CASE NUMBER		SIZE IN MILLIMETERS										
		1	2	3	4	5	6	7	8	9	10	10+
1	neg.	2	0	5	4	2	3	2	0	0	2	0
	pos.											
2	neg.	2	3	12	13	10	6	1	1	1	0	0
	pos.			**1**	**7**	**8**	**5**	**6**	**1**	**1**	**1**	**5**
3	neg.	1	3	2	2	4	0	1	0	0	0	0
	pos.				**1**	**1**		**2**				
4	neg.	4	10	15	7	6	3	0	2	1	2	1
	pos.				**2**							
5	neg.	5	13	13	10	9	2	4	7	1	4	2
	pos.											
6	neg.	7	14	22	7	7	1	0	0	0	1	0
	pos.				**1**			**1**				
7	neg.	2	20	6	10	1	0	0	0	0	0	0
	pos.											
8	neg.	8	15	3	8	6	2	0	0	0	0	0
	pos.				**1**							
9	neg.	5	8	9	9	5	4	2	3	2	0	3
	pos.				**1**			**1**	**2**			**1**
10	neg.	4	14	12	7	9	3	0	1	1	1	1
	pos.											
11	neg.	14	8	15	6	3	1	0	0	0	0	0
	pos.											
12	neg.	0	13	20	9	3	2	0	0	0	1	0
	pos.											
13	neg.	2	4	3	2	0	0	0	0	0	0	0
	pos.					**2**		**1**		**1**		
14	neg.	12	16	14	4	5	2	0	0	0	0	0
	pos.			**1**	**4**	**6**	**1**	**1**	**1**		**1**	
15	neg.	1	19	20	11	3	0	0	0	0	0	0
	pos.				**1**							
16	neg.	3	3	3	3	1	0	0	0	0	0	0
	pos.				**2**	**2**	**1**	**2**	**2**			**1**
17	neg.	20	18	11	2	0	0	0	0	0	0	0
	pos.											
18	neg.	11	30	7	2	0	0	0	0	0	0	0
	pos.			**7**	**8**	**2**	**2**	**4**		**2**	**1**	**2**
19	neg.	27	35	21	4	1	4	1	0	0	0	0
	pos.				**2**			**2**			**1**	
20	neg.	15	21	13	7	1	1	1	1	0	0	0
	pos.			**2**	**3**	**3**	**2**					
Totals	neg.	145	267	226	127	76	34	12	15	6	11	7
	pos.			**11**	**33**	**24**	**11**	**20**	**6**	**4**	**4**	**9**

a significant number of tumor emboli if only every fifth, tenth, or twentieth section is examined? This question can and should be settled, first, by an actual examination of serial sections of the lymph nodes and, second, by an application of the laws of probability governing such an examination in a mathematical analysis.

SUMMARY

Our evaluation of the Spalteholz method as applied to the study of the lymph node metastases in cancer of the rectum has shown it to be a satisfactory, but time-consuming, procedure. Our data are in agreement with those obtained by others. However, 2 questions arising directly out of this study remain unanswered. What is the relationship between the size of a lymph node and the incidence of metastasis in it? How many sections should be prepared

of each lymph node in order to find all of the tumor emboli? From our data, the size relationship appears to be a crucial one. An effort is being made to find satisfactory answers to these questions in further investigations.

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A NEW OPERATIVE APPROACH TO INFLAMMATORY STRICTURES OF THE RECTUM AND RECTOSIGMOID

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THE purpose of this report is to describe a new operative solution to the problem of the patient who has extensive inflammatory strictures of the rectum and rectosigmoid. This consists of an abdominoperineal excision of the diseased large bowel, preservation of the sphincter, and closure of the gap between the descending colon and the sphincter with a length of ileum swung down on its mesentery. A case is reported in which this procedure has been done with re-establishment of normal fecal movements through the anus and with complete postoperative continence.

INTRODUCTION—THE PROBLEM

Lymphogranuloma venereum is a venereal disease that is not uncommon in many parts of the United States. Treatment in the past has been largely symptomatic, consisting of chemotherapeutic agents that reduce secondary infection, but which, unfortunately, do not directly attack the virus. As a result, many individuals afflicted with this disease develop anorectal fissures, fistulas, abscesses, and finally dense fibrous strictures. Such strictures usually arise in that portion of the rectum just above the anal sphincter, but often extend proximally and may even involve the entire left colon.[6] Carcinomatous degeneration of such involved areas of bowel may be more frequent than formerly realized, having been reported as high as 8.6 per cent by some authors.[2, 8, 22]

The treatment of lymphogranulomatous strictures has consisted of various drugs, irradiation, dilatation, and various forms of surgical extirpation. As stated above, chemotherapeutic agents and various antibiotics in the past have only controlled secondary infective organisms. Now that aureomycin and chloramphenicol are available, we have weapons that seem to attack the etiological agent directly. Such therapeutic agents will not, however, affect the firm fibrous adhesions that represent the end result of the untreated infection.

Irradiation has not proved to be of any real benefit.[22]

Dilatation has had ample trial, and it will, of course, allow many patients with minimal strictures to remain in relative comfort as long as dilatations are continued. The patient who returns year after year for dilation in the outpatient Rectal Clinic is well-known evidence that such a procedure is effective but not definitive. More vigorous dilatation, under anesthesia, is often attempted but with very mediocre results and with a surprising percentage of complications, including peritonitis, overwhelming sepsis, and death.[6, 22]

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The obvious way to deal with an uncontrolled inflammation or stricture of the rectum is by colostomy. This is the simplest attack. In a number of cases this is adequate, for the inflamed area is put at rest. Most cases requiring colostomy, however, are in an advanced state and, unfortunately, many patients do not benefit from this procedure. In Woods' series at the Cincinnati General Hospital, for example, there were poor results in 60 per cent of the severe cases in which colostomy was performed. Only 17.8 per cent of this group of 28 patients had what he termed a good result.[22] Those experienced in treating lymphogranuloma venereum point out that the diseased and infected bowel remains, sometimes bleeding, often causing abscesses, fistulas and unpleasant discharge. Occasionally, carcinoma may develop.[6, 9]

Excising the stricture through the sphincter and pulling the rectum down to the perianal skin is a direct approach to the problem.[8, 23] In the occasional case this is adequate, but in more extensive lesions it will not suffice since diseased bowel may remain above the point of resection.

The Lockhart-Mummery combination of colostomy and perineal excision has been given ample trial.[23] Edwards and Kendell reported 6 cases treated in this way with a good result in 5 of the patients.[6] They and others admit that they cannot predict how high the ulceration and stricture formation may extend. The same objection can be raised to other modifications of the sacroperineal approach.

The next step, that of abdominoperineal resection according to the Miles technique, has been approached warily. As Bacon[1] stated in 1941, ". . . any maneuveur which calls for division of the peritoneal floor at the time the stricture is extirpated carries a mortality that triples and even quadruples similar operations for carcinoma, namely 65 to 80 per cent." Barber and Murphy[2] at Bellevue Hospital treated 35 of the 55 patients with lymphogranulomatous stricture on the Surgical Service by abdominoperineal resection. The experience of these authors has been similar to that of Woods and Hanlon[22] at Cincinnati—namely, that in a large percentage of cases such patients are relieved from the local and systemic effects of the disease.[2, 6, 11, 22]

The obvious drawback to abdominoperineal resection is that of colostomy. Although a very small price to pay for survival and cure in malignant disease, it assumes inflationary proportions when dealing with a benign inflammatory disease. Since the anal sphincters often are uninvolved and since wide dissection around the perianal area is not necessary in the benign condition, sphincter saving is feasible and colostomy avoidable.

In order to accomplish this end and to bridge the large gap between the colostomy and the sphincter, the operative procedure described below was devised.

CASE REPORT

History.—Mrs. L. E. at the present time is a 53-year-old Negro woman. In 1924, she was first admitted to the Barnes Hospital, where a rectal stricture was dilated and a fistula in ano excised. She was followed in the Washington University Clinics, and in

1927, a supravaginal hysterectomy, right salpingo-oophorectomy, left salpingectomy, and appendectomy were performed. In 1928, multiple fistulas in ano were excised. In 1929, dilatation of rectal strictures under anesthesia was undertaken. In 1935, a Frei test was found to be positive and the diagnosis of lymphogranuloma venereum made. In 1936, left oophorectomy and drainage of a pelvic abscess was performed. A suprapubic urinary fistula resulted but finally closed spontaneously. At this time a high sigmoid colostomy was performed proximal to the involved large bowel. In 1948, mild cardiac failure on a hypertensive basis was noted and she was digitalized. Later, in 1948, the colostomy was revised.

During the past 10 years or more she has repeatedly pleaded to have the colostomy closed. Examination in the Rectal Clinic showed almost complete closure of the rectum by a dense fibrous stricture just inside the sphincter. The sphincter was intact and of good tone. There was no evidence of active infection.

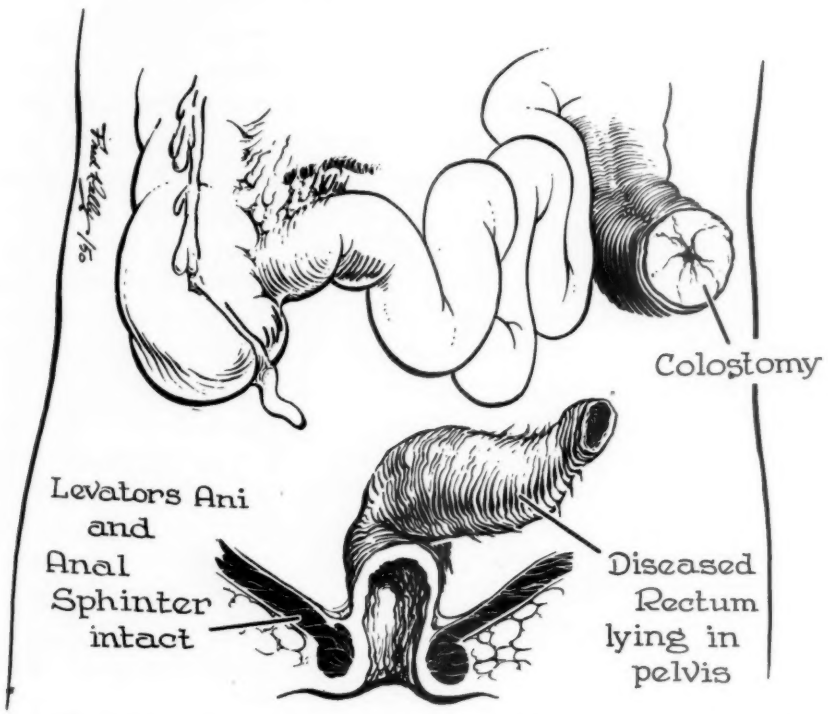

Fig. 1.—Condition prior to excision of the diseased rectum and rectosigmoid colon. Dense fibrous adhesions completely stenosed the lower rectum and extended up to the colostomy.

She was admitted to the Barnes Hospital on Feb. 14, 1950, for evaluation of a plastic procedure in an attempt to re-establish anal defecation.

On Feb. 21, 1950, the first stage of the plastic repair was undertaken. On the anterior abdominal wall were many old scars resulting from previous operations and fistulas. A lower midline incision was made and the peritoneal cavity entered. A tremendous number of dense fibrous adhesions were encountered, but no evidence of inflammatory activity was noted. The distal loop of the colostomy was identified and the scarred and stenosed sigmoid colon and rectum visualized (Fig. 1). The colostomy was a high one, and it was impossible to mobilize the left colon sufficiently to pass it into the pelvis. Since there was no indication for wide resection, it was possible to carry the abdominal and pelvic dissection close to the bowel wall. Dissection from above was carried to the levator ani muscles. The terminal ileum was then transected at 2 points about 25 cm. apart, thus forming an isolated mobile segment of bowel (Fig. 2). This point was chosen since it

was the lowest lying segment of small bowel and offered a minimum amount of difficulty in mobilization for the pull-through procedure. An end-to-end anastomosis was made between the cut ends of the ileum, and the rent in the mesentery was closed. The freed segment of ileum was then mobilized on its mesentery, with its blood supply being carefully preserved.

Attention was then directed to the perineum, and the patient was put up in lithotomy position. The abdomen was left open so that work could be carried on simultaneously from above and below. Since the stricture lay immediately inside the rectum, there was no attempt to save a cuff of rectal mucosa. Dissection was carried out from within the anal canal, the mucosa was incised circularly at the anorectal junction, and the dissection was carried through all layers of the rectum. Once outside the rectum proper, dissection proceeded superiorly until communication was established with the dissection from above. The rectum and rectosigmoid colon were then pulled through the sphincter and removed.

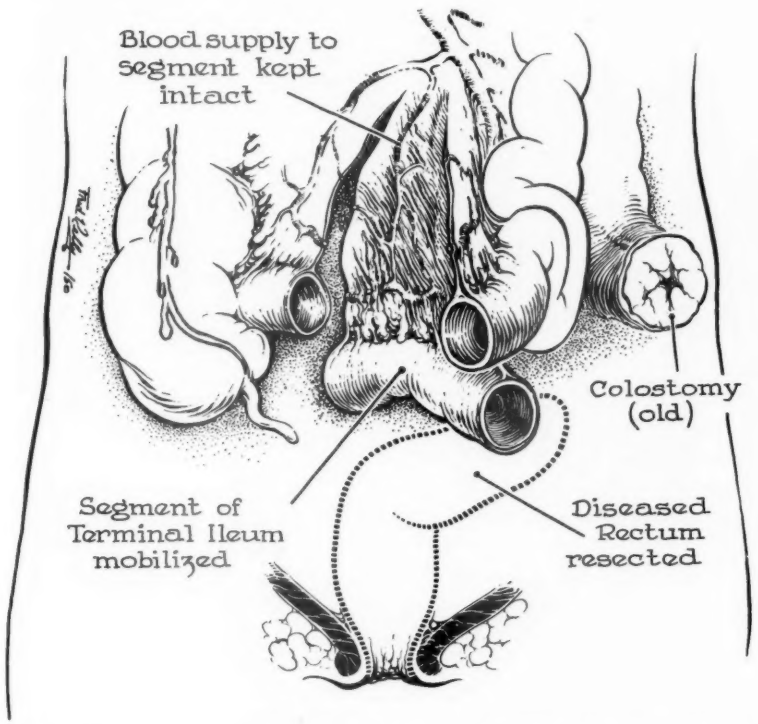

Fig. 2.—The abdominoperineal resection is completed, but with the anal sphincter intact. A 25 cm. segment of ileum is mobilized prior to swinging it down into the pelvis.

The distal end of the ileal segment was then threaded down into the pelvis through the hole in the peritoneum to the levator ani muscles. The ileum was grasped from below and brought out through the sphincter. The ileal mucosa was then sutured to the perineal skin. No drain was inserted. Before the patient was taken out of lithotomy position, the blood supply and viability of the terminal portion of the ileum were assured.

The remaining portion of the operation consisted of approximating the levator ani muscles and peritoneum to the ileum transplant. The ileal mesentery lay adjacent to the posterior peritoneum and was sutured in place to prevent internal herniation. The abdomen was closed in the usual way, bringing the proximal segment of the transplanted ileum out through the incision just adjacent to the left lower quadrant colostomy.

Postoperatively there were no complications. Aureomycin had been given 2 days preoperatively and was continued for 6 days postoperatively. A "rectal" examination

was done on the tenth day and the sphincter found to be of good tone. Proctoscopic examination showed normal ileal mucosa. The patient was then discharged from the hospital for a 2 week period.

The last stage of the operation was carried out on March 24, 1950, and consisted merely of an intraperitoneal anastomosis of the colon to the proximal end of the ileal segment (Fig. 3). The difference in the diameters of the 2 portions of bowel was less than anticipated and by incising the ileum obliquely the disproportion was eliminated.

Postoperatively there were no complications. On the fourth postoperative day the patient began to pass feces through the ileal segment. Within a week the stools were formed and at no time was she incontinent. She was discharged on the eighth postoperative day, passing stools through the anus for the first time in 15 years.

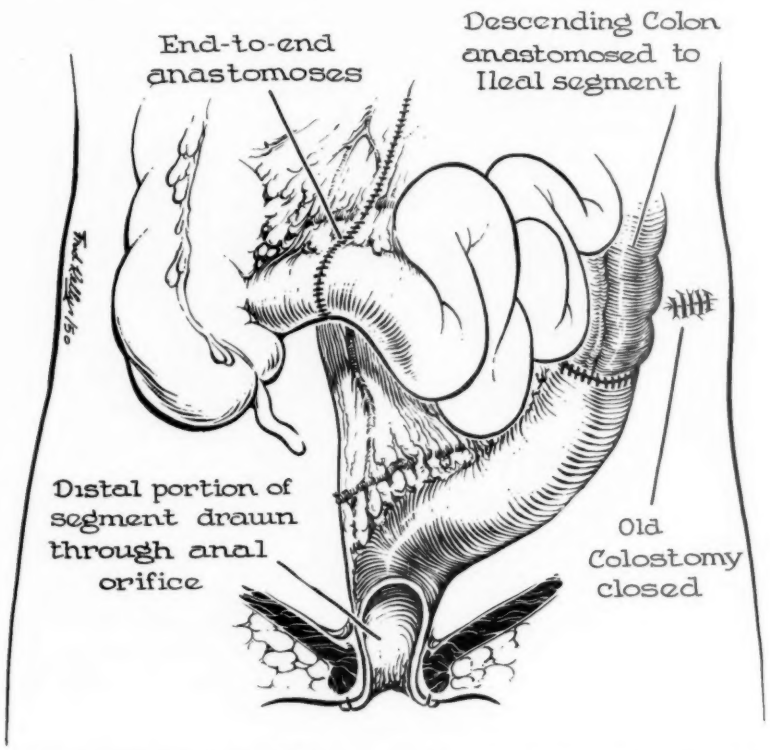
End-to-end anastomoses
Descending Colon anastomosed to Ileal segment
Distal portion of segment drawn through anal orifice
Old Colostomy closed

Fig. 3.—The operation is completed. Fecal continuity has been re-established.

Her postoperative course has been most interesting. Although it has been only 10 months since the final operation, this patient states that she has but 1 or 2 movements per day. The movements are formed and, more important, she is completely continent. She does not soil herself at night and states that she has attended a 3 hour church service as well as a double-feature movie without any discomfort or fear. Nine months after operation an adhesive band in the right lower quadrant was severed which had been causing some symptoms of low-grade intestinal obstruction. The anastomosis sites were well healed. Since this time she has continued to do well.

A barium enema x-ray examination taken 2 weeks after the final operation showed a normal small bowel pattern up to the site of anastomosis with the descending colon (Fig. 4).

The patient states that the desire to defecate is somewhat different from what she remembered 15 years ago prior to her colostomy. She says that the stool seems to be held "rather high up," then passes down quite easily and is expelled. She is able to control the stool once it has passed down into the lower segment, but says that it requires some

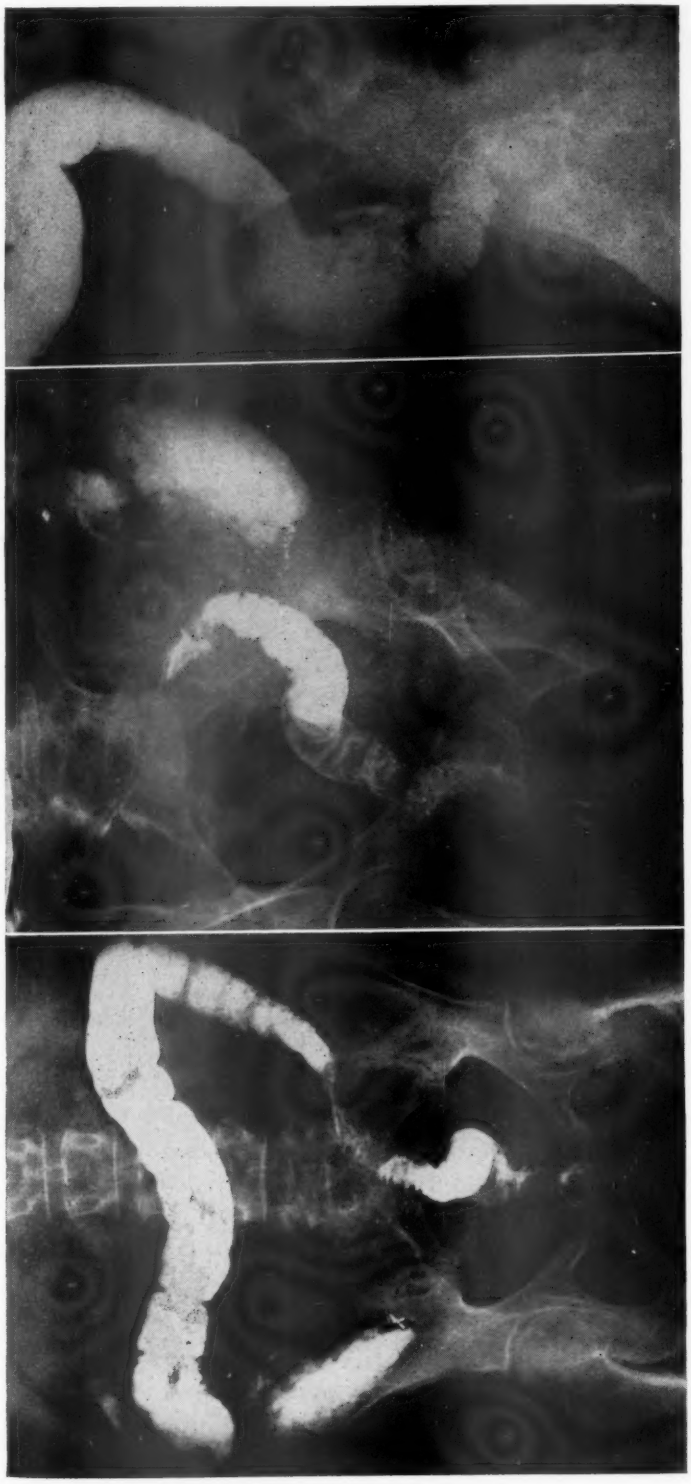

Fig. 4.—Barium enema x-ray examination 2 weeks after completion of the operation. Note the normal small intestinal pattern from the large bowel anastomosis to the perineum and also the early dilatation of the ileum just proximal to the sphincter.

small degree of concentration. She has upon request held the stool for as long as one-half hour just inside the sphincter.

In an evaluation of this patient it must be remembered that she is of good intelligence and for 15 years previously had accustomed herself to a colostomy.

DISCUSSION

Before carrying out this procedure on the patient just described, Mueller and Fischer[10] had completed a similar operation on 4 dogs. Working in the surgical laboratories of the Washington University Medical School, they had anastomosed an ileal loop proximally to the descending colon and distally to a stump of rectum. The distal perineal anastomosis was similar to the technique employed by Swenson and Bill.[19]

In reviewing the literature on this subject it was found that a number of European authors had explored the possibilities of coloileorectoplasty. Crespi,[5] in 1890, substituted a piece of ileum for a segment of descending colon in several dogs. In 1910, Vignolo,[20] in Pisa, carried out such a procedure on a patient with carcinoma of the descending colon. Fenwick,[7] in England, used Murphy buttons on both ends of a segment of ileum and thus spliced together the ends of the descending colon. Thereafter, the procedure was used by Quenu,[16] Picot,[15] Okinczyc,[13] and Pauchet.[14] Stone[18] recorded the only case in the American literature.

All of these procedures were designed to join 2 ends of large bowel together by bridging the gap with ileum. Most of the procedures were carried out inside the peritoneal cavity, though Vignolo[20] and Quenu[16] both did their anastomoses below the peritoneal reflection.

The technique herein described differs from previous procedures in that the pull-through technique of Ravitch and Sabiston[17] is used with the distal segment of ileum and no distal anastomosis is employed.

It is of interest that several authors, using ileal segments as connecting links in the large bowel, obtained histologic studies of the interposed small bowel. They found that the ileum hypertrophied and assumed many of the microscopic characteristics of the adjacent colon.[5, 14, 20]

Indications for such an operative procedure are, of course, limited, but in the occasional patient may be of benefit, as illustrated by the case described. Benign lesions of the left colon, sigmoid, or rectum that make a colostomy inevitable offer the ideal indication for this procedure. The candidate for such a procedure must, of course, have a functioning anal sphincter. In some cases where a large gap exists between the left colon and rectum, the large bowel can be mobilized and approximated to the distal stump. When, however, the bowel will not reach to the intact sphincter, this pull-through procedure is of no benefit.

Surgical manipulation in lymphogranuloma venereum has, as we have seen, been fraught with danger in the past, for no chemotherapeutic agent or antibiotic has been available to attack directly the specific virus. Now that such agents are available, the scope of both extirpative and reparative surgery can be increased with a greater sense of security.

SUMMARY

1. A short résumé is presented of the surgical treatment of rectal strictures due to lymphogranuloma venereum. The tendency toward radical excision of lymphogranulomatous strictures by abdominoperineal resection is noted.

2. A case is presented in which a sphincter-saving abdominoperineal resection was done for an advanced lymphogranulomatous stricture. A new operation is described wherein a length of ileum is swung on its mesentery to connect the left colon with the intact sphincter. The entire diseased rectum and sigmoid colon are thus excised, leaving the patient with a normal, controllable, completely continent anal fecal aperture.

3. It is noted that several surgeons have in the past used ileal segments to bridge gaps in the left colon, but none has used the pull-through technique herein described.

4. Indications for such a procedure are briefly outlined.

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FATAL COMPLICATION OF THE MIKULICZ OPERATION AND THE MONT REID PROCEDURE FOR PROCIDENTIA OF THE COLON AND RECTUM

I. L. Lichtenstein, M.D., Los Angeles, Calif., and A. W. Ulin, M.D., Philadelphia, Pa.

TWO deaths following operative intervention for sigmoidal procidentia[1] emphasize the possible dangers arising from the use of either the Mikulicz operation[2-4] or the Mont Reid procedure.[5, 6]

Case 1 (H. H. 16732).—J. K., a 67-year-old white man, was admitted on Sept. 24, 1948, with the chief complaint of massive protrusion of the clostomy. In February, 1948, a sigmoid loop colostomy was made in the left lower quadrant of the abdomen for obstructing carcinoma of the rectum with metastases. Subsequently, it was necessary to resect the prostate through the urethral route for urinary retention. It was during an attempt to void that the patient first noticed the procidentia. This became considerably more pronounced and was such a great annoyance that he insisted on a repair.

On physical examination, the condition of the patient was judged to be generally good. Nothing remarkable was noted in the proximal loop of the colostomy. There was a complete procidentia of the distal loop for approximately 18 cm. (Fig. 1). This was reducible, but with removal of pressure, the segment immediately prolapsed. It seemed an ideal case for the Mikulicz operation. Accordingly, the entire procidentia was transected slightly above the skin level. The serosa of the intussusceptum was united to the serosa of the intussuscipiens with interrupted silk sutures. A second layer of continuous chromic catgut through all the layers of both walls was then inserted (Fig. 2). The suture line was replaced into the lumen of the distal colostomy loop. In effect, then, a primary end-to-end anastamosis in the distal sigmoid loop was accomplished, after resection of the prolapsed segment.

Three days postoperatively, blood was noted coming from the distal colostomy. A finger inserted into the stoma palpated an intact anastamosis. The patient became progressively more distended and gradually presented well-developed peritonitis with clinical icterus. Despite supportive measures, the patient died fourteen days following surgery. Post-mortem examination revealed a segment of gangrenous and perforated sigmoid colon, distal to the stoma, with multiple intraperitoneal and hepatic abscesses. A main branch of the left colic artery and the marginal artery to the distal sigmoid had been compromised by the operative procedure.

Case 2 (H. H. 6354).—W. D., an 80-year-old white man, was admitted on Feb. 4, 1948, with a chief complaint of rectal hemorrhage, severe for one day. He was chronically constipated, and the bowels had not moved for four days prior to his admission. Cascara catharsis was administered at home on Feb. 3, 1948, with resultant strain, massive hemorrhage, and protrusion of a rectal mass.

Upon examination, it was felt that this mass represented a complete rectal prolapse with strangulation of the intussusceptum. At its apex this segment had an ulcerating, hemorrhaging lesion, later proved to be carcinoma. There was no evidence that small bowel was involved. The patient was in shock.

Measures to combat shock were instituted. A scout film of the abdomen failed to reveal any evidence of small bowel obstruction. Because of the patient's age and critical condition, immediate operation was considered ill-advised. Furthermore, the strangulated bowel and perforated malignant lesion discouraged the hope of any approach by laparotomy. Accordingly, a large rectal tube was inserted into the rectum and two heavy strangulating

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Fig. 1.—Procidentia, distal loop of sigmoid colostomy.

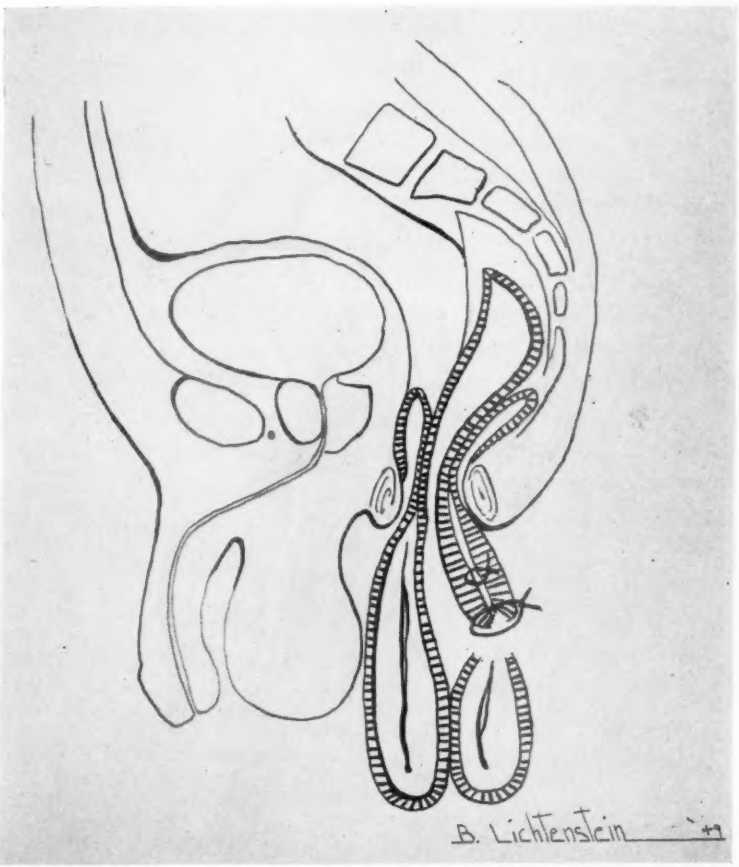

Fig. 2.—Mikulicz operation for procidentia of rectosigmoid colon. This technique was applied in the first case.

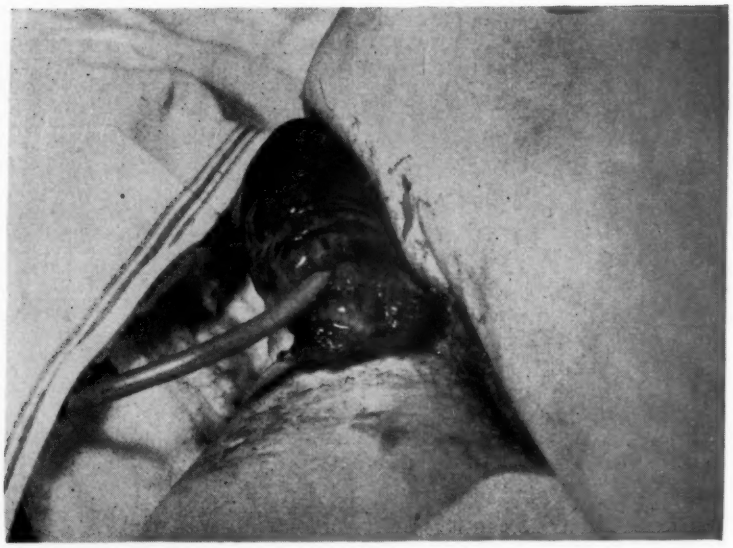

Fig. 3.—Mont Reid procedure in second case. Note ulcerated carcinoma at apex of the intussusceptum.

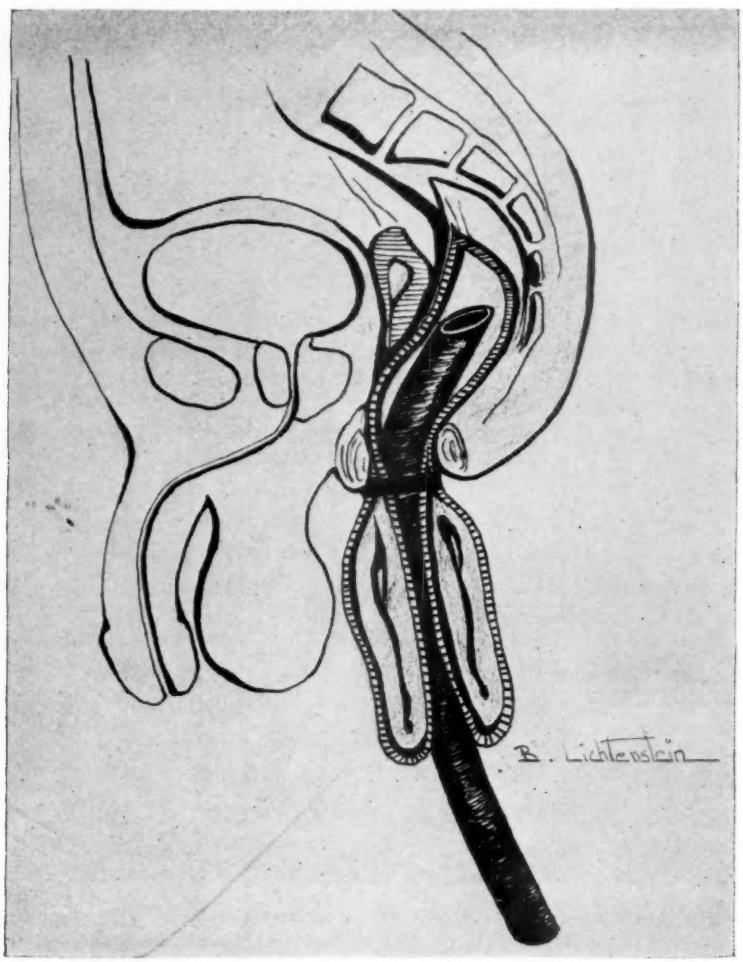

Fig. 4.—Diagrammatic portrayal of situation as it existed in second case, revealed at necropsy.

silk ligatures were applied to the neck of the procidentia, according to the method of Mont Reid (Fig. 3). In this way it was hoped to control the loss of blood from the strangulated complete prolapse. In that respect, the treatment was successful. The patient made a remarkable recovery from a precarious state.

Three days after admission, it was noted that the patient had suddenly developed a frank peritonitis and was distended, despite the fact that the rectal tube was draining. There was no vomiting. All conservative measures failed and the patient died on his fourth hospital day.

Autopsy revealed that in addition to the sigmoidal intussusception, the slippage of the rectum and its supporting structures had apparently caught a loop of proximal redundant sigmoid colon in the rectovesical pouch. This loop was pinched off, much as in a Richter's hernia (Fig. 4). There was no complete obstruction of the lumen, but the post-mortem examination revealed the involved portion of bowel to be gangrenous and perforated, with frank peritonitis.

DISCUSSION

Many types of operations have been suggested for procidentia of the colon and rectum, one of which is the Mikulicz type operation, that is, transection of the intussuscepted bowel. It is the purpose of this paper to caution against this method, since this "not very serious" operation may be dangerous, or even fatal. Actually, the procedures used in the two cases reported have much in common. Anatomically they produce the same result and avoid laparotomy. However, in the Mikulicz procedure, the peritoneal cavity is opened and may be examined about the operative site. Nevertheless, it is impossible to properly evaluate the vascular tree. Peritoneal contamination is more likely than in the Mont Reid method, where an eroding ligature causes an inflammatory agglutination of the peritoneal surfaces. In the latter however, a hernial sac is being ligated without first being opened to reveal the nature of its contents. In the second case, it was an unsuspected internal hernia that proved the undoing of an apparently beautifully indicated and applied procedure.

It is true that extenuating circumstances may sometimes influence the choice of procedure. With poor-risk carcinomatous patients, in one case of which hemorrhage was a vital factor, it could be argued that abdominal exploration was contraindicated if bleeding could be otherwise controlled. However, today, with improved anesthesia, availability of blood transfusions, antibiotics, and so on, there is ordinarily little excuse for blind operations. Direct inspection of pathologic anatomy in its normal relationships has no parallel.

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A NEW METHOD FOR OBTAINING BARIUM ENEMAS IN COLOSTOMY PATIENTS

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INTRODUCTION

ALTHOUGH the site of the original lesion cannot be demonstrated, follow-up barium enema studies are important in patients who have had an abdominoperineal excision of a carcinoma of the rectum.

Mayo[2] reported in 1942 that of 334 patients who died with primary carcinoma of the colon or rectum, 114, or 34 per cent, had polyps in addition to the primary neoplasm. Fourteen per cent of these polyps were found to be malignant. In 131 of the 334 cases resection of the rectum or colon had been done as a curative procedure. In this latter group death occurred within 3 weeks of the operation, and in 49 cases, or 37 per cent, residual polyps were found. Benson and Berger[1] in 1945 reported that in a series of 344 patients with carcinoma of the large bowel collected over a 10-year period 16, or 4.6 per cent, had multiple malignant growths in this organ. In 5 of the patients the second carcinoma was found at 1, 1½, 7½, 10, and 11 years after the original growth was removed surgically. In the remaining 11 cases the second or third carcinoma was found at the same time or within 9 months of the original lesion. These authors also stated that 84 consecutive autopsies on patients who had died of carcinoma of the large bowel showed one or more polyps to be present in 21, or 25 per cent.

In order to facilitate barium examinations in colostomy patients, various devices such as the bulb of an asepto syringe[3, 4] and Bardex catheters[5] have been used to retain the barium suspension in the residual colon during the examination. However, there are drawbacks to these methods. During the examination, barium often leaks around the bulb of the catheter, running over the abdominal wall and over the x-ray equipment. In addition, during the period of evacuation and while the postevacuation films are being taken, emission from the colostomy cannot be controlled and there is further soiling of the patient and equipment. This causes much embarrassment to the patients who as a group are very fastidious about their colostomies and pride themselves in the degree of care and control they have attained. Previously, the procedure has been so disagreeable that we have had patients refuse to report back for further follow-up examinations.

METHOD

The equipment required for the method of examination to be described is very simple and is pictured in Fig. 1. It consists of a Rutzen type ileostomy

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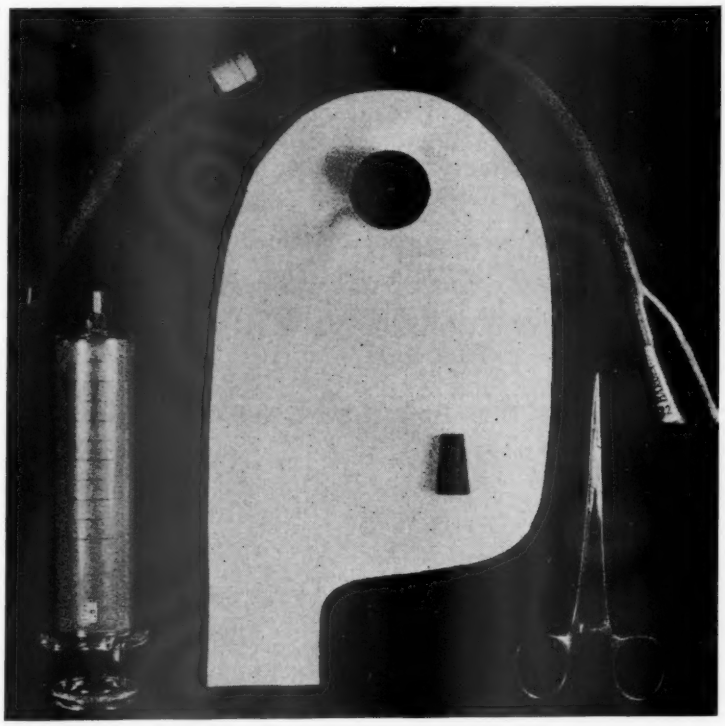

Fig. 1.—Basic equipment necessary for doing barium enemas in patients with colostomies.

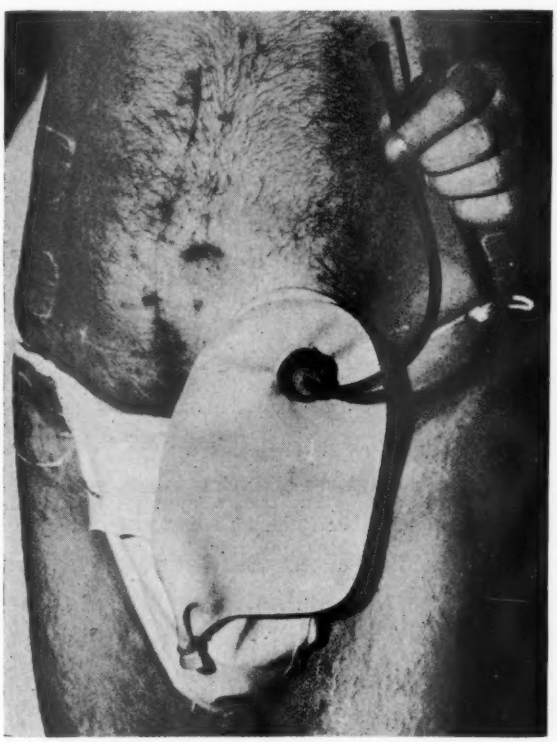

Fig. 2.—Ileostomy bag applied.

bag, a No. 22 Bardex catheter fitted with a stopper, several corks, a 50 c.c. syringe, and a hemostat. The hole in the face plate of the ileostomy bag is enlarged to about 2.5 cm. in diameter in order to fit the colostomies which are, in general, larger than ileostomies. Directly opposite this hole, another one is cut in the bag 1.5 cm. in diameter into which a No. 6 black rubber cork is inserted and firmly tied. This cork is grooved around the edge to aid in fastening it to the bag, and it contains a hole in the center large enough to receive the stopper which is on the Bardex catheter. This stopper is placed about 12 cm. from the tip of the catheter, but may be adjusted to correspond to the thickness of the abdominal wall.

Fig. 3.—Postevacuation film showing good filling of the entire colon and of the terminal ilium. Note that residual barium in the ileostomy bag does not interfere with the details of the colon.

The patients are given the usual instructions preparatory to a barium enema so that they come for the examination with the colons empty. The skin about the colostomy is cleaned well with benzene, facing cement is applied to the face plate of the ileostomy bag, and skin cement to the skin about the colostomy. The cement is allowed to dry until "tacky." With the Bardex catheter fitted snugly in the cork which is tied in the front of the bag, the bag is applied over the colostomy guiding the tip of the catheter into the colostomy. Fig. 2

shows the ileostomy bag in place. For use on obese patients the brass face plate of the ileostomy bag may be snapped so that it has a slightly concave surface rather than convex, which is the usual situation. In addition a belt that is not radiopaque may be worn to give further support to the bag, but is not necessary. The procedure of applying the bag should take not more than 5 minutes.

With the patient on the fluoroscopy table a preliminary check of the abdomen may be made, after which the Bardex bag is blown up to a maximum of 15 c.c. of air. The bag at this inflation measures 2.7 cm. in diameter, which is not enough to injure the gut, yet is enough to prevent the catheter from slipping out because the hole in the face plate is less than this diameter. There is no leakage of barium around the balloon even at this low inflation because the pressure of the barium suspension in the colon seats the balloon against the face plate of the Rutzen bag with the thickness of the abdominal wall intervening.

The free end of the Bardex catheter is attached to the source of barium which may be run in at any speed desired, and the fluoroscopic examination is carried out with no sense of urgency due to fear of leakage. After the initial films are made a hemostat is clamped across the catheter and the patient taken to the nearest bathroom. The air is released from the Bardex balloon, the catheter is removed from the bag, and a small cork inserted in its place. The rubber band is removed from the bottom of the Rutzen bag, which is allowed to hang into the toilet bowl so that any barium that is emitted into the bag necessarily drops into the waste receptacle. Fifteen to 20 minutes are required for this initial evacuation. The bottom of the bag is again occluded and the patient returned for the postevacuation films. Any barium leaking during this stage of the procedure is retained in the bag until the examination is over. If desired, air contrast studies may be done. The residual barium in the bag does not interfere with the outline of the colon, as is shown in Fig. 3. When all the films are taken, the patient is allowed to return to the toilet to evacuate the colon further until he thinks it is safe to remove the bag, put on his own device if he wears one, and return home. The equipment is easily cleaned for further use and actually could be put up as a kit by a central supply room or by a technician.

COMMENT

Thirty patients have been examined by this method to date, and satisfactory fluoroscopic examinations and x-ray films have been obtained in all cases. It has been found to be reasonably quick, and the situation is under control at all times. Some examinations have been obtained that otherwise would have been impossible. Several facts of importance have become apparent, however. If there is marked scarring about the colostomy causing deep fissures, especially in fat people, difficulty is encountered in making the bag stick for the lack of a flat surface. In these patients a belt should be worn to give the bag extra support. At no time in any patient should the bag be allowed to become overdistended and heavy with fluid for it will pull away from the skin, causing leakage.

SUMMARY

1. The need for adequate follow-up examinations in patients with excision of the rectum or lower colon for carcinoma is stressed.

2. A simple method for obtaining barium enemas in colostomy patients is outlined.

3. Thirty patients have been studied and satisfactory examinations have been obtained, avoiding embarrassment to the patients and inconvenience to the personnel.

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EXTENSIVE RESECTION OF THE SMALL INTESTINE FOLLOWED BY VAGOTOMY

Experimental Study of Fat and Nitrogen Excretion on Dogs

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OUR experience with patients requiring extensive resection of the small bowel has stimulated our interest in this problem.[1] It is a helpless feeling to watch an otherwise healthy individual starve because his intestine is too short to furnish adequate nourishment from ingested food. The problem of fat absorption in postoperative surgical patients, however, has a much wider application than this. Disturbances of fat absorption with consequent inadequate nutrition are frequent, especially following surgery of the gastrointestinal tract. Means of improving intestinal absorption and nutrition are of much interest to the surgeon and of possible benefit to the surgical patient.

Extensive resection of the small intestine in man is followed by a variable and unpredictable response depending upon the length of the bowel resected, the length and health of the remaining segment of the small bowel, and probably other factors which are not clear at present. The procedure is usually followed by diarrhea, weight loss, and sometimes anemia, especially in the severe cases. The changes may be severe and lead to death.[1] On the other hand, the changes may be consistent with life though making the patient weak and undernourished. At still other times, extensive resection apparently is tolerated with very little demonstrable effect.[2-18]

Though the effect varies in intensity in different individuals, in general, a shortened transit time develops with bulky, fatty stools, weight loss, sometimes anemia,[19] and, in severe cases, inanition and death.

Jensenius[20] reported less disturbance following resection of the proximal portion of the small bowel in dogs than compared to resection of the distal portion.

Antispasmodic drugs and low-fat, high-caloric diets have been of benefit in many cases. Dennis and associates[21] have reported prolongation of the transit time in the small bowel following vagotomy in man. Cannon[22] early reported prolongation of transit time in cats following section of the vagus nerves. Faik, Grindlay, and Mann[23] have recently noted decreased intestinal activity at various levels of the small bowel but these changes were not comparable in degree to those occurring in the stomach following vagotomy.

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Reasoning that a prolongation of the transit time might be favorable to the absorption of fat from the shortened intestine, the following experiments were carried out on dogs. An attempt was made to answer the following questions: (1) Is the transit time through the shortened bowel of the dog following resection significantly prolonged by vagotomy? (2) Is the absorption of fat and nitrogen as well as the general nourishment of the animals altered following vagotomy? (3) Is there a significant difference between the resection of the proximal small bowel as compared with resection of the distal small bowel?

GENERAL PROCEDURE

Full-grown mongrel dogs weighing 7 to 18 kg. were maintained on a low-fat kennel diet (7.8 per cent fat, 5.2 per cent nitrogen). All absorption studies, however, were carried out using the high-fat diet suggested by Jensenius[20] (23 per cent fat and 6.4 per cent nitrogen). This was done in order to make our work comparable, as near as possible, to the work of this author. Determinations were made by absorption experiment on the per cent of fat and nitrogen and the per cent of ingested fat and nitrogen excreted in the stool during the last 3 days of a 5 day absorption study. Glucose tolerance tests were also done.

Two-thirds of the small bowel was then resected. Approximately one-half the animals had the proximal portion of the small bowel removed starting just distal to the bile ducts, and the other one-half had the distal two-thirds of the bowel removed. Intestinal continuity was re-established by end-to-end anastomosis for the distal resections, and side-to-side anastomosis for the proximal resections. After several weeks, absorption studies were repeated and the amounts of fat and nitrogen excreted in the stool determined. These determinations were carried out periodically over a one-year period. The transit time of barium from the stomach to the cecum was also determined, as well as the gastric emptying time in some animals. Sometime during the year, transthoracic vagotomy was carried out on the animals and the changes in transit time and fat and nitrogen excretion determined, as well as changes in gastric emptying time. Three animals were observed for 5 months following resection before the vagus nerves were cut.

Insulin hypoglycemia tests were performed following vagotomy on all animals and repeated at the end of one year. In all animals, repeated tests indicated that the vagotomy was complete even at the end of the experimental period.

METHODS

Operations.—All operations were performed under intravenous Nembutal anesthesia using a right rectus incision for laparotomy. The small intestine was measured from pylorus to cecum along its mesenteric border, using umbilical tape, and a resection of two-thirds of the small bowel was performed. Transthoracic vagotomy was performed sometime after bowel resection through the fourth or fifth rib bed on the left and 4 cm. segments of each nerve were resected. Intratracheal positive pressure anesthesia was employed for the transthoracic vagotomy in each instance.

Absorption Experiments.—After they had fasted 24 hours, the dogs were fed the Jensenius diet in an amount of 20 Gm. per kilogram for 4 days. Feces passed during the first 2 days were discarded. All stools were collected daily for the following 3 days. The final stools were collected 24 hours after the fourth feeding. Attempts to mark the stools with lamp black and ferrous sulfate were not satisfactory. Specimens were kept in sealed containers and analyzed for fat and nitrogen content soon thereafter or frozen until analysis. Total fat was determined by a modified method of Fowweather, and nitrogen by the Kjeldahl method. In the instances of vomiting or poor appetite, the material was weighed back before the next regular feeding.

Glucose Tolerance Tests: Oral glucose tolerance tests in the fasting state were done using 2 Gm. of glucose per kilogram (20 per cent solution) administered by stomach tube. Blood sugar determinations were made every 15 minutes for 2 hours thereafter. Probably this amount of glucose is too great. Better curves would probably have been obtained with 1 to ½ Gm. of glucose per kilogram of body weight.[24]

Insulin Hypoglycemia Tests: These tests were carried out according to the method described by Hollander.[25] Regular insulin in an amount of 1 unit per 3 kg. body weight was used intravenously.

Transit Time and Gastric Emptying Time: Following intestinal resection and following vagotomy at various times, the speed with which barium passed through the intestine from stomach to cecum was determined. After a 12 hour fast, 50 Gm. of barium sulfate in thin suspension was introduced into the stomach by tube and its passage from the stomach through the intestine followed by means of serial roentgenograms taken at 10 to 15 minute intervals. The time for the barium to reach the cecum was determined, and on a number of animals the time required for the stomach to empty was also measured.

RESULTS

Proximal Resections.—Fig. 1 gives the results in 3 animals which had the proximal two-thirds of the small bowel removed. This shows graphically the changes in the per cent of fat and nitrogen in the stool, and the per cent of fat and nitrogen excreted in Dogs 1, 3, and 4 following resection and vagotomy 5 weeks later. These animals normally had an average of 11.5 per cent fat in their stools and excreted an average of 3.5 per cent of their ingested fat. Following resection, the average per cent of fat in the stool rose to 40.5 per cent, and the average per cent of fat excreted was 12.5 per cent. However, following vagotomy, it is seen there was a drop in the per cent of fat in the stool as well as the excretion of the ingested fat. These figures, in fact, returned nearly to normal 6 weeks after vagotomy and remained down for 19 weeks. During this time, we were encouraged, thinking we had possibly found a method of increasing fat absorption. However, it is noted that, though the figures remained low for several months, there was a gradual rise in fat excretion as time went on. Fifty-three weeks after resection there was 44 per cent fat in the stool and a fat excretion of 26 per cent. These figures were somewhat lower 7 weeks later. The

alterations in protein absorption are seen to be very much less, as shown by the lower 2 lines on the graph. The per cent of nitrogen in the stools rose slightly immediately after resection. The rise in excretion of ingested nitrogen was much less than the rise in excretion of ingested fat. It appears from these results that the changes in protein absorption following extensive resection of the small bowel are much less than those for fat. It can also be said, in general, that the least amount of fat and nitrogen excretion occurred when the transit time through the small bowel and the gastric emptying time were longest.

The weight changes in the animals are represented by the upper line of the graph (Fig. 1). It is seen that the average weights expressed in per cent of the

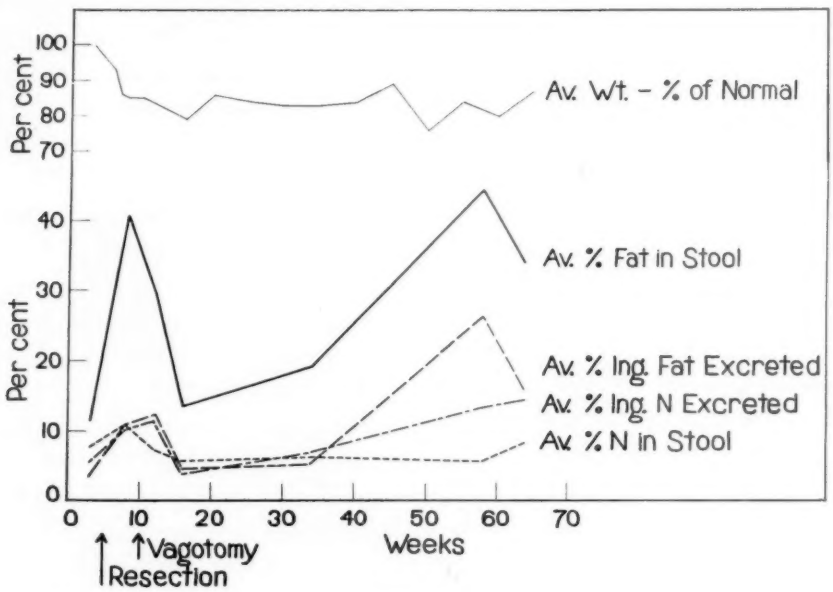

Fig. 1.—Average values for 3 dogs with resection of proximal two-thirds of the small bowel. Vagotomy was carried out 5 weeks after resection.

preoperative weight fell to 86 per cent 2 weeks after resection. The curve flattens at this level with a further drop to 79 per cent on the fifteenth week, 5 weeks after vagotomy. At no time does the weight rise significantly above the prevagotomy level. The animals are in a delicate balance and are prone to develop diarrhea, weight loss, and anemia. Such a period is shown by the drop in the weight curve to 76 per cent on the fiftieth week.

Distal Resections.—Fig. 2 gives the average results for animals 6, 7, 8, and 9 after resection of the distal two-thirds of the small bowel. The changes are seen to follow the general trend of the proximal resections for both fat and nitrogen excretion. The increase in the per cent of fat in the stool immediately following resection was not as marked in the dogs with distal resection as in those with proximal resection. A temporary diminution in the excretion of fat and nitrogen in the stool is noted following vagotomy as with the proximal resections. This diminished excretion of fat again persisted for about 5 months, though the weights of the animals did not improve. However, at the end of

one year, there seems to be no significant difference between the proximal and distal resections in regard to the utilization of fat and protein. The dogs with distal resection had an average of 42 per cent fat in the stool and were excreting approximately 20 per cent of their ingested fat. The proximal resection dogs, on the other hand, had 44.5 per cent fat in the stool and were excreting 26 per cent of the ingested fat when fed the Jensenius diet. The last determinations on the proximal resections show a reduction of the amount of fat in the stool. However, if the last 2 determinations for each group are averaged, the proximal resections have 39 per cent fat in the stool and excrete 21 per cent of the ingested fat while the distal resection animals have 43.5 per cent in the stool and excrete 21 per cent of the ingested fat. These figures, in our opinion,

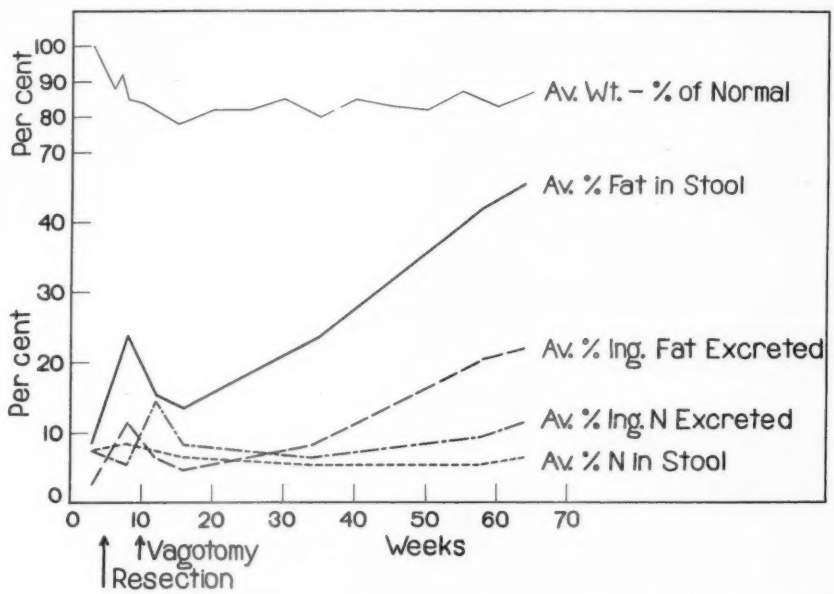

Fig. 2.—Average values for 4 dogs with resection of the distal two-thirds of the small bowel. Vagotomy was carried out 5 weeks after resection.

indicate no significant difference between resection of the proximal small bowel as compared to resection of the distal small bowel. This is substantiated by the nutritional response of the animals, as well as survival rates in the 2 groups. The average weights of the animals expressed in per cent of the preoperative weight is shown in the upper curve of the graph (Fig. 2). The animals lost an average of 16 per cent of their body weight during the first 5 weeks following resection. The transthoracic vagotomies caused an additional transient weight loss of 6 per cent. The lowest average weight of the 4 dogs occurred 5 weeks after vagotomy.

Late Vagotomy.—Having noted the decrease in fat excretion in the previous 2 groups of animals beginning 2 to 3 weeks after vagotomy, and 7 to 8 weeks after bowel resection, we decided to observe resected animals over a longer period of time to determine whether or not this might be a spontaneous change following resection alone.

The results in Dogs 11, 13, and 16 are shown in Fig. 3. Of these 3 animals, one had a proximal resection and two had distal resections. The intestines were resected as with the earlier animals and the changes in fat and nitrogen excretion observed over a period of 22 weeks. Transthoracic vagotomy was then carried out and further observations were made.

It is seen in Fig. 3 that the per cent of fat in the stool and the per cent of ingested fat excreted rose sharply as it did in the previous 2 groups of animals following resection. The curve for stool fat is particularly similar to the first part of the stool fat curve in Fig. 1. Ten weeks following resection, there is a drop in stool fat to 34.2 per cent. However, the excretion of ingested fat rose to 25 per cent, which is higher than the average figures after early vagotomy for either the proximal or distal resections. Though there was diminution in fat excretion as shown on the twenty-first week of the experiment, the figures did

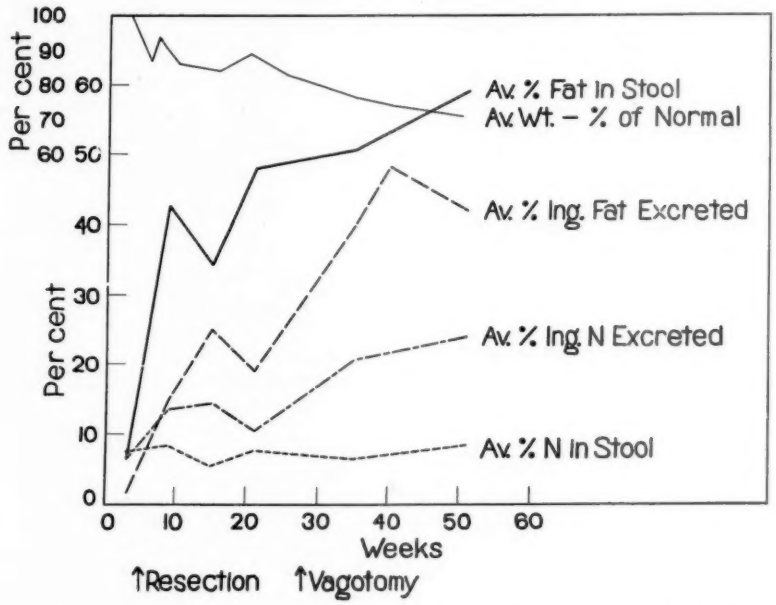

Fig. 3.—Average values for 3 dogs which received vagotomy 23 weeks following resection of two-thirds of the small intestine.

not return to the normal range as they had following bowel resection and early vagotomy. Furthermore, following vagotomy in the twenty-eighth week of the experiment, there was no diminution in fat excretion. In fact, fat and nitrogen excretions were increased and the animals showed an additional weight loss which was maintained following vagotomy, as shown in the upper curve of the graph.

Changes in Transit Time and Gastric Emptying Time.—In the animals with the proximal portion of the small bowel removed, the average time required for the head of the barium column to pass from the stomach to cecum was 97 minutes, 4½ weeks after resection. Three weeks after section of the vagus nerves the average transit time was 120 minutes, and at the end of one year this figure had decreased to 87 minutes or to essentially the same figure as before vagotomy.

In the animals with the distal portion of the small intestine removed, the average transit time from stomach to cecum was shorter than with the proximal resections. Four weeks after resection, the average time required for barium to pass from stomach to cecum was 43 minutes. Five weeks after transthoracic vagotomy, the transit time was 45 minutes, 10 weeks after vagotomy, 75 minutes, and at the end of the one year back to 65 minutes.

In the 3 animals that received late vagotomy, the average transit time from stomach to cecum through the shortened intestine 3 weeks after resection was 37 minutes. Eight weeks after operation, the average time was 51 minutes. Transthoracic vagotomy was carried out during the twenty-second postresection week. Three weeks after vagotomy, the average transit time was 63 minutes, and 21 weeks after vagotomy, the average transit time from stomach to cecum was 65 minutes, somewhat longer than before vagotomy.

These changes are not great, yet it is difficult to know how important a few minutes may be in the absorption of fat from the bowel. The animals with the proximal portion of the small bowel removed had a longer transit time than those with the distal portion removed. The general nutritional state and survival rate, however, was the same in the 2 groups.

The other more marked change following section of the vagus nerves was great prolongation of the gastric emptying time. This prolongation caused food to enter the shortened small intestine over a very much longer period of time. The average gastric emptying time normally was 1 to 3 hours. One week after vagotomy, the stomachs were large and atonic, and frequently contained barium 24 hours after its administration. Four weeks after vagotomy, the gastric emptying time ranged from 10 to 12 hours. At the end of 1 year, the gastric emptying time was still within this range. As noted by Faik, Grindlay, and Mann,[23] the changes in gastric emptying time were greater than the changes in transit time through the small bowel following vagotomy.

Alterations in Blood Picture.—Martin, Robertson, and Dennis[19] observed the anemia which may follow massive small bowel resection in the dog to be of an inconstant type. These authors resected all but 8 to 10 inches of jejunum and ileum and observed anemia in 4 animals which lived a period of several months. The changes in color index were not constant but no definite trend toward macrocytosis could be established. The anemia was considered to be a result of the marked state of malnutrition which developed.

Our animals had less extensive resections than those of Martin and associates and were observed over a longer period of time (one year or more). Seven of the 12 animals developed anemia as months went by. Four of these had proximal resection, and three had distal resection. In all cases, anemia was either normochromic or hypochromic. Macrocytosis was observed in none of our animals. These findings agree with the shorter term experiments of Martin, Robertson, and Dennis.

It was noted that the animals with extensive bowel resection were in a delicate nutritional balance. They were easily thrown into a state of diarrhea and weight loss. If this was allowed to progress, anemia and hypoproteinemia developed, and which, if not corrected, was a serious prognostic sign.

Animals That Did Not Survive.—Seven animals died following resection during the course of or at the close of the experiments. One of these had an ileo-ileal intussusception with gangrenous bowel 3 weeks after resection and another died of peritonitis from a leaky anastomosis on the third postoperative day. Three other animals died of inanition. They were unable to absorb sufficient nourishment from the shortened intestines to remain in a good state of health. Two of these had proximal resections of the small bowel and died 2 and 4½ months after resection, and did not have their vagus nerves sectioned. The fifth animal had a distal resection of the small bowel and vagotomy 23 weeks later. It remained in a very poor state of nutrition but seemed to be holding its own until the forty-third week, when it began having loose stools. It developed extreme weight loss down to 48 per cent of the preoperative weight and died of inanition on its fiftieth week after resection. The sixth animal died of a perforated gastric ulcer (Fig. 4) 57 weeks after resection of the distal two-thirds of the small bowel and 33 weeks after vagotomy. The seventh animal died of pneumonia 18 months after resection and 17 months after vagotomy. The distal portion of the stomach contained 6 gastric ulcers 3 to 6 mm. in diameter.

Fig. 4.—Stomach of a dog which died of perforated gastric ulcer 57 weeks following resection and 33 weeks following transthoracic vagotomy. Three animals have died following section of the vagus nerves. Two of these have had gastric ulcers.

Glucose Tolerance.—Oral glucose tolerance test showed, in general, a flat curve following resection of the proximal two-thirds of the small bowel with the blood sugar frequently at its highest level at the end of 2 hours. This agrees with the accepted idea that glucose is absorbed high in the gastrointestinal tract. Following resection of the distal portion of the small bowel, no appreciable change in the glucose tolerance curves could be detected. We could detect no significant change in the form of the glucose tolerance curve following vagotomy regardless of the segment of bowel resected.

Utilization of Fat on High and Low Fat Diets.—Considering the comparatively good health of our animals at the end of one year, balance studies were made on the animals on the kennel diet and these results compared with those of the animals on the high-fat Jensenius diet.

Table I shows the results of this study. The dogs ate the Jensenius diet poorly but ate the kennel diet very well, so that the average amount of fat ingested was essentially the same for the three day experimental period (103 to

TABLE I. UTILIZATION OF FAT AND PROTEIN ON HIGH AND LOW FAT DIETS

DOG	TOTAL GM. DIET 3 DAYS	TOTAL GM. FAT	TOTAL GM. N	GM. FAT EXCRETED	PER CENT FAT STOOL	PER CENT FAT EXCRETED	PER CENT N STOOL	PER CENT N EXCRETED	DIET	
4	512	118	33	17.4	28.7	15	6.8	12.7	Jensenius	
7	404	93	26	10.7	30.1	11.6	3.8	53	Nitrogen	6.5%
9	456	106	29	11.7	37.4	11	—	—	Fat	24%
11	418	96	26	14.3	45.2	15	6.1	16		
Average	446	103	28		35.3	13.1	5.6	11		
4	1455	113.5	76	5.3	5	4.7	10.7	14.7	Kennel	
7	1131	88.2	59	1.91	1.7	2.1	10.3	19	Nitrogen	5%
9	1377	107.4	72	1.81	1.25	1.6	9.8	12.5	Fat	8%
11	1395	108	73	4.0	3.2	3.7	11	18		
Average	1339	104	70		2.8	3	10.4	16		

104 Gm. fat). On the Jensenius diet, the stools contained an average of 35 per cent fat and an average of 13 per cent of the ingested fat was excreted. On the low-fat kennel diet, the stools contained an average of 2.8 per cent and an average of 3 per cent of the ingested fat was excreted, which is a normal figure. There was higher loss of nitrogen on the kennel diet, but this was not great.

We feel that this explains the comparatively good health of most of the animals at the end of the year, though their utilization of fat on the high-fat test diet was poorer than it had been shortly after resection. This substantiates the findings that patients with extensive resection also do best on a low-fat diet.

DISCUSSION

Following resection of two-thirds of the small bowel, there was a marked rise in the excretion of ingested fat and a slight rise in excretion of ingested nitrogen. The change in absorption of ingested sugar shown by the glucose tolerance curves was not great; the curves were flattened following resection of the proximal portion of the small bowel. It appears that extensive resection of the small bowel shows its greatest effect by changes in the excretion of fat in the stool. This would seem due to the lack of ease with which fat is absorbed.

The reactions of the animals to resection of the proximal small bowel were not significantly different from those following resection of the distal small bowel with the exception of somewhat less shortening of the transit time and a flatter glucose tolerance curve following proximal resection. No difference in survival rate of either group of animals was noted and the fat and nitrogen excretions were found to be essentially the same in each.

The mechanism by which fat absorption was increased by early vagotomy and not affected by late vagotomy remains unexplained. Fox and Grimson[26] reported defective fat absorption in 8 of 9 patients who had distress following vagotomy. These patients, however, did not have fat absorption studies prior to vagotomy.

The fact that transit time was somewhat prolonged following vagotomy might explain the diminution in fat excretion in the early vagotomy cases following proximal and distal bowel resection. It would seem, however, that a similar effect should have occurred in the dogs which had vagotomy late following bowel resection; however, this was not the case, suggesting that other factors are involved. The effect of gastric emptying time falls into a similar category. It would seem the prolongation of gastric emptying time following vagotomy might well be favorable to increased fat absorption since the stomach contents are discharged slowly into the shortened intestine over a long period of time.

However, the changes in gastric emptying time were the same following late vagotomy as with early vagotomy and no beneficial effect on fat excretion was noted with the former.

The presence of gastric ulcers in 2 of the animals which died near the end of the experiment is of much interest. The complication of gastric ulcer following vagotomy has been noted previously.[27-32] These authors observed gastric ulcers in 7 to 60 per cent of animals following section of the vagus nerves. Maingot[33] reported 3 cases of gastric ulcer in 44 human cases of vagotomy for duodenal ulceration.

The fact that each of the 7 animals that developed anemia over a period of months developed a hypochromic anemia is of interest, and it is interesting that no animals developed macrocytic anemia.

SUMMARY

Mongrel dogs were subjected to resection of two-thirds of the small intestine. In one group of animals the proximal portion of the small bowel was removed, and in another group the distal portion of the small bowel was removed. The changes in fat and nitrogen excretion in the stools were studied for one year after resection and oral glucose tolerance tests were done before and after operation.

At various times after resection (5 to 23 weeks) transthoracic vagotomy was done on the animals. Transit time, gastric emptying time, and fat and nitrogen excretion in the stools were followed for approximately one year.

Transit time through the remaining small bowel segments was only slightly altered by section of the vagus nerves and in some animals this was not permanent.

Gastric emptying time was greatly prolonged after vagotomy and persisted to the close of the experiment more than one year later.

Early vagotomy had no permanent effect on fat and nitrogen excretion. It caused temporary improvement during the time when gastric emptying and transit time were most prolonged. No improvement in nutrition was noted, however, during this interval of diminished fat and nitrogen excretion.

No significant difference was detected between animals which lost the proximal portion of the small bowel and animals which lost the distal portion of the small bowel except that glucose tolerance curves were altered more when the proximal small bowel was removed and transit time was somewhat slower.

Late vagotomy had no beneficial effect and was probably detrimental to the animals.

Seven of the 12 animals developed anemia several months after two-thirds resection of the small bowel. In all cases, this was a hypochromic anemia. Macrocytosis was not observed.

Two animals autopsied 17 and 33 weeks following vagotomy showed gastric ulceration, one of which was perforated.

CONCLUSIONS

1. Late vagotomy following extensive resection of the small intestine has no beneficial effect on the dog and is probably detrimental.

2. Early vagotomy following resection has no permanent effect on fat and nitrogen excretion in the stool. Temporary improvement in fat and nitrogen excretion was noted but no improvement in nutrition.

3. Transit time through the shortened small bowel was not greatly prolonged following transthoracic vagotomy. Gastric emptying time was greatly prolonged up to the close of the experiment more than a year later.

4. No significant differences were noted between animals which had resection of the proximal portion of the small bowel as compared to the distal portion. Transit time was somewhat slower following proximal resection and the glucose tolerance curves were flatter. Fat and nitrogen excretion, general nutritional state and survival was essentially the same in the two groups of animals.

5. The animals utilized a high carbohydrate, low fat diet best.

6. Anemia, when it developed, was of normochromic or hypochromic type.

7. Gastric ulceration occurred in 2 of 3 dogs which died 4 to 8 months after vagotomy.

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TRANSPLANTATION OF THE ILEUM

An Experimental Study

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ATTEMPTS to remove sigmoid and rectal lesions with preservation of the anal sphincter parallel the development of modern surgery. The efforts of Kraske[1] and Hochenegg[2] have been continued to the present day, new procedures being devised, older procedures being reviewed and modified. Despite this intense activity, the problem is not always clearly defined, nor are the indications or results always accepted. The approach to this subject must begin with the consideration of diseases of the colon and rectum, and their inherent differences. Problems pertinent to carcinoma are not identical to those present in inflammatory disease or other pathologic processes.

It is the purpose of this paper to propose an alternate method for restoring continuity of the large bowel using an interposed loop of ileum, and preserving the anal sphincter following removal of long segments of rectum and colon. This procedure could be of use in malignant, as well as nonmalignant, conditions of the bowel. The initial problem in malignant lesions of the bowel concerning the restoration of continuity and preservation of sphincter is often confused. There is a furor in the surgical literature concerning removal of the rectum distal to the carcinomatous growths. Buie[3] estimated retrograde spread of carcinoma to be in 1 per cent of cases. Glover and Waugh[4] found nine cases out of 100 with retrograde spread, eight with 2 cm. spread and one with 6 cm. caudal invasion by the tumor. The primary problem in the attack of malignant neoplasm is adequate resection of lymph node bearing tissue proximal or cephalad to the lesion. Buie said, ". . . almost the entire spread as far as metastasis is concerned is cephalad." Sugarbaker[5] recommended, ". . . all the inferior mesenteric node chain should be removed for tumors in the upper rectum and rectosigmoid area." An excellent discussion of the blood supply to the sigmoid and rectum in relation to radical resection of long segments of bowel and extensive removal of lymph node bearing mesentery with the saving of the sphincter and restoration of continuity is made by Goligher.[6] He stated that one should ". . . apply the main ligature roughly opposite the bifurcation of the abdominal aorta" in the combined abdominoperineal resection of Miles. "If [anterior] resection operations are to compete with combined excision, it is essential that in them, also, ligation should be carried out at this point and no lower, and rather than compromise on this issue, the attempts at [anterior] resection should be abandoned in favor of a combined excision." Goligher con-

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demned the sacral resection operations on this basis, and in addition was critical of the low ligations mentioned in the published reports of Wangensteen,[7] Bacon,[8] and Muir[9] in their procedures for restoring continuity and preserving the sphincter. He stated that "Ligation of the inferior mesenteric vessels may be accomplished exactly as in a combined excision and yet sufficient sigmoid colon preserved for re-establishing continuity," and went on to outline clearly his treatment of individual vessels and their variations to accomplish this. However, he realized that, in some cases, sacrifice of the greater part of the sigmoid may be necessary, leaving inadequate length for an anastomosis, and in these cases he recommended either mobilization of the transverse colon or splenic flexure and descending colon, allowing the bowel to be brought down into the pelvis, or the utilization of a loop of ileum between the colon and rectum as we are advocating. Bacon mentioned ten cases in which the former maneuver was performed without a death.

Due to a fixation of tissue by inflammation in nonmalignant disease, primary anastomosis cannot always be performed to restore continuity. It is in such nonmalignant conditions that the procedure proposed is thought to be more useful than it is in malignant disease. A clinical report of the successful use of this proposed procedure in resection of the rectosigmoid and rectum for lymphogranuloma inguinale is the subject of a subsequent report by Eiseman and Mueller.[15] A more complete discussion of the problems pertinent to one particular inflammatory condition will be presented there.

It is realized that this herein proposed procedure is not new. Fenwick,[10] Stone,[11] and Wangensteen[7] have suggested similar procedures. Our work was not stimulated or suggested by these earlier papers but by the recent work of Ravitch and Sabiston,[12] and by Swenson and Bill.[13] Ravitch devised an anal ileostomy with preservation of the sphincter for patients requiring total colectomy for nonmalignant disease. Swenson and Bill's procedure of resection of the rectum and rectosigmoid with preservation of the sphincter was utilized for spastic segments of colon and rectum producing megacolon.

EXPERIMENTAL WORK

Mongrel dogs, male and female, weighing from 20 to 55 pounds, were operated upon by the following techniques. Preoperative preparation consisted of Sulfathaladine 2 to 4 Gm. per day for three to five days. During this time dogs were fed fresh meat and dog biscuits until the day before operation, when only milk was given. After being given a dose of castor oil by stomach tube, they were fasted 12 to 16 hours preoperatively. Intravenous Nembutal, 25 mg. per kilogram, was the anesthetic used.

The abdomen and perineum were shaved and then prepared with iodine and alcohol. Aseptic technique was used throughout. No supportive therapy was given during operation. Duracillin, 200,000 units per day, was given intramuscularly for one to three days postoperatively and the postoperative diet consisted of milk and water the first few days, followed by regular kennel diet of fresh meat and dog biscuits.

OPERATIVE PROCEDURES

A lower midline abdominal incision was made. The cecum was visualized and a loop of terminal ileum, preferably one with two arteries supplying it, was isolated by cutting the mesentery back to its root and dividing the bowel at each end. The continuity of the ileum was then restored by an open end-to-end ileoileostomy with two rows of 0000 chromic catgut, and the mesentery was approximated with a running stitch of fine catgut. A rubber-shod clamp was left on the proximal end of the isolated ileal loop, while the distal end was occluded securely with a length of heavy silk (Fig. 1). The sigmoid colon was then mobilized and, after placement of rubber-shod clamps, divided. The proximal end of the ileal loop was swung over and anastomosis between it and the descending colon was carried out with 0000 chromic catgut. In some instances, the ileal

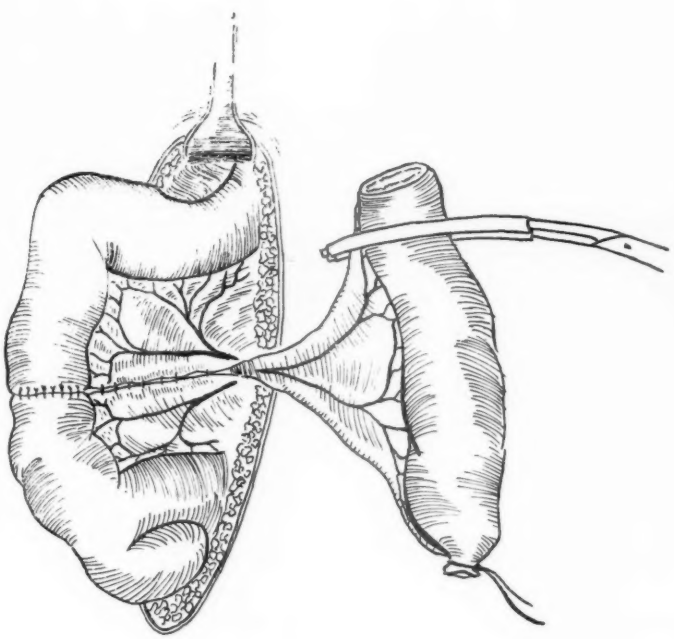

Fig. 1.

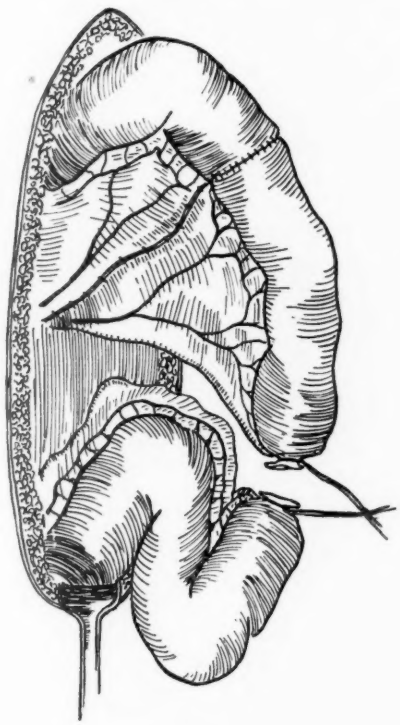

Fig. 2.

Fig. 1.—Diagram showing isolation of ileal segment on its mesentery and end-to-end anastomosis of the ileum.

Fig. 2.—Diagram showing anastomosis of descending colon to the proximal portion of the transplanted ileum. The distal end is tied to the upper end of the rectum to be pulled through the everted anus.

loop was opened a few millimeters on the antemesenteric border to make the two ends involved in the anastomosis more nearly equal. The mesenteries of the ileal loop and the descending colon were then approximated with fine catgut down to the base of the mesentery of the descending colon.

The mesentery of the sigmoid colon was incised and the rectum freed down to the anus by sharp and blunt dissection. After hemostasis was obtained, the heavy silk ligature of the distal end of the ileal loop was secured to another

heavy silk ligature on the upper end of the sigmoid to be resected (Fig. 2). These two loops (the ileal transplant and the completely mobilized sigmoid) were replaced in the curve of the sacrum. Care was taken to carry the abdominal-pelvic dissection down into the internal sphincter, dividing all pelvic attachments of the rectum, to allow the perineal stage to be done without difficulty. No attempt was made to repair the pelvic peritoneum. The abdominal wall was then closed in layers with fine and medium silk.

The perineal procedure was that of Swenson and Bill with a slight modification: a clamp was inserted into the rectum, the rectum grasped and everted. This everted mucosa with surrounding perineal field was prepared with iodine and alcohol and a circular incision made through the full thickness of the bowel wall at the anorectal junction. The rectum and sigmoid, now completely freed except for the ligature attaching it to the ileal loop, were pulled completely out of the pelvis; the ileal loop followed and presented at the everted anus. The musculature coat of the ileum was sutured to the internal sphincter and the heavy ligature and its crushed cuff of ileum were excised. The ileal mucosa was sutured to the anal mucosa and the protruding bowel then replaced into the pelvis.

In two dogs, a loop of ileum was isolated in the manner just described. A short segment of sigmoid was then resected and the loop of ileum interposed between the descending colon and rectum, a descending colon-ileum anastomosis and an ileum-rectum anastomosis being performed with 0000 chromic catgut in two layers.

Stool size, consistency, and frequency, and general health and nutritional state of the dogs were observed. Dogs surviving the operative procedures were sacrificed six to ten months following operation, and autopsy was performed. Preparations were examined and measured in the gross, and photographs were taken. Histologic sections were made of ileum functioning as colon and rectum.

RESULTS

A total of nine dogs were operated upon, four of which survived for sufficient length of time for adequate observation. The following points should be noted:

Three animals with replacement of the rectum survived for relatively long periods, enjoying good health, and were observed sufficiently long to evaluate stool habits. One dog passed through an extended period of anal incontinence and then became continent. Two other dogs were not completely continent, though they had formed stool. The dog which was continent was observed four months longer (ten months in all) than the two dogs not completely continent. It may be that a considerable length of time is needed for continence to be established. This may depend on the length of time required for the ileal loop to take on the characteristics and function of the large bowel, resulting in more well-formed stool. As soon as more solid stool is formed, the sphincter may increase in efficiency.

It is to be noted, too, that five of the nine dogs died early of peritonitis. The cause of the peritonitis in two of these was leakage at the sigmoido-ileal

anastomosis. One dog died secondary to peritonitis and abscess at the ileo-ileostomy. A fourth dog died due to a gross technical error at the time of operation when the heavy occlusive ligature slipped from the rectum, allowing feces to contaminate the pelvis. The fifth had a peritonitis with no demonstrable leak.

One dog (CR-13), in which ileum was transplanted into the sigmoid, died ten weeks postoperatively of an intussusception of the ileo-ileal anastomosis into the cecum, dragging along the mesentery of the loop transplanted into the sigmoid and causing vascular occlusion to this segment.

Examination of this specimen (Fig. 3) showed that the ileum had taken on gross characteristics of colon with large rugae and an increase in circumference. There was no evidence that any blood supply to this portion was derived from adjacent colon across the lines of anastomosis. The bowel was too necrotic to permit microscopic examination of the interposed ileum.

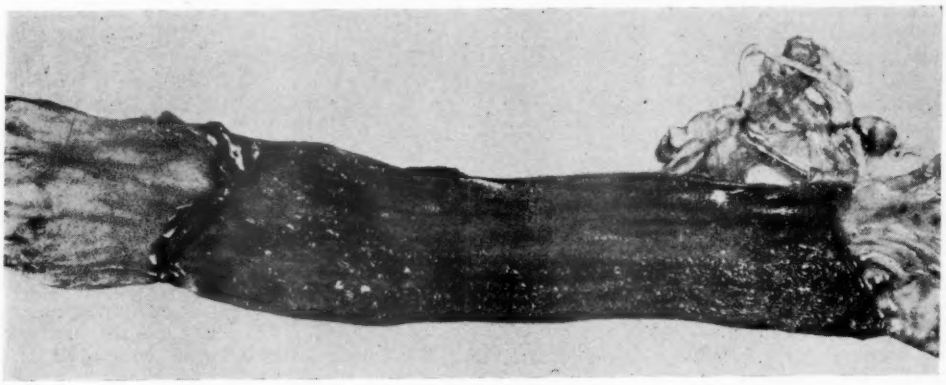

Fig. 3.—Post-mortem photograph of segment of ileum transplanted into the sigmoid. Intussusception of the mesentery of this segment caused vascular occlusion and gangrene.

This operative procedure must be assessed on several separate points: operative mortality, long-term survival in good health, ability of ileum to substitute as colon, and continence of the sphincter. The high immediate mortality of the series of dogs should not detract from the value of the operation. The four dogs surviving, and all but one of those dying postoperatively, had viable ileal loops. One death in the group is explained by an abscess at the ileo-ileostomy site (CR-6), another by a gross technical error or massive contamination of the pelvis by feces during operation (CR-8). Failure of the sigmoido-ileal anastomosis occurred in the Dogs CR-5 and CR-12. This difficulty could have been lessened by the establishment of a double-barreled colostomy composed of sigmoid and ileum instead of the primary anastomosis of bowel. In fact, this was advocated and performed in one clinical case (Eiseman and Mueller). In only one case was the anorectal stump nonviable, and this was in a case in which a long rectal stump inadvertently was left, in a dog which developed peritonitis. In clinical cases better pre- and postoperative care would be a matter of course. Antibiotic therapy, supportive fluids, electrolytes, blood and nutrients, and post-

operative intestinal suction would greatly lower the mortality in the human being. Our mortality figures are inferior to those of Swenson, but our procedure consisted of three separate intestinal anastomoses rather than one.

The ability of the ileum to function as colon has been demonstrated by Quenu[14] and others. In these animals, stools were at first loose but later became firmer and less soft. It is evident that a loop of interposed ileum will not keep the feces too soft. The transplanted ileal wall undergoes structural changes in time.

Grossly, the ileum dilated to a diameter almost equal to that of the descending colon. The mucosa showed coarse, thickened, longitudinal rugae which resembled colon to some extent (Fig. 4). Microscopic examination of the ileum, however, showed fairly normal ileal architecture. There was an increase in the number of polymorphonuclear leucocytes, but no striking alteration in size of the villi, thickness of muscle or number of mucoid cells.

Fig. 4.—Post-mortem photograph of segments of ileum transplanted between descending colon and anus.

The most controversial question is that of the ability of the anal sphincter to function following an operative procedure of this type. Complete continence was obtained in one of the three dogs surviving extensive resections and anastomosis of the ileum to anus. The preliminary evidence indicates that continence may take a long time to develop, possibly depending on the changes in the ileum allowing formed stool rather than liquid stool to be passed. In any procedure involving this much manipulation of the sphincter, a long period of time should also be allowed for the sphincter to regain its tone. Swenson reported normal bowel control in his surviving dogs. There is no difference in the interference with the function of the sphincter itself in Swenson's procedure or ours. The difference lies in the consistency of the stool arriving at the sphincter, and this may be overcome in time by changes in the ileal loop.

SUMMARY

1. An alternative method for restoration of continuity of the lower bowel with preservation of the sphincter following extensive resections of rectum and colon is presented.

2. The possibilities for use of the transposed ileal loop in malignant and nonmalignant disease are discussed.

3. Results of this operative procedure in dogs indicate that continence may be obtained after a period of time, though only 1 cm. of rectum remains.

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EXPERIENCES IN EXPERIMENTAL PORTACAVAL ANASTOMOSIS

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ANASTOMOSIS of the portal vein to the inferior vena cava has become definitely established in recent years as a treatment for portal hypertension with bleeding from esophageal varices. The anastomosis may be performed in two ways: either end-to-side, with the end of the portal vein to the side of the inferior vena cava, or side-to-side between the ventral side of the inferior vena cava and the dorsal side of the portal vein. The relative merits of the two types of anastomosis have not been definitely settled. The side-to-side type has been considered to be more physiologic, as some blood can continue through the liver. This may well be of some importance, as, according to the basic work of Fishback,[1] the regeneration of lobes of the liver after partial hepatectomy has been performed is proportional to the hyperemia occurring in the portal vein. The postulates upon which this statement is based are summarized by Markowitz[2] as follows:

1. Restoration of the liver does not occur following partial hepatectomy if an Eck fistula has been established previously, either with or without ligature of the portal vein above the anastomosis.

2. Birds have a natural Eck fistula in the form of the circle of Jacobson and partial hepatectomy is not followed by restoration. However, restoration occurs if the anastomotic vessel is ligated.

3. If one branch of the portal vein is tied in the normal animal, the corresponding half of the liver atrophies and the other half enlarges, in a manner typical of hepatic restoration. However, it has been our opinion that, although this work proves the importance of portal pressure in hepatic regeneration, it is not necessarily of significance, as the quantity of pressure remaining in the portal vein after side-to-side portacaval anastomosis may not be sufficient to have any effect on the regeneration of the liver.

In the end-to-side type, all of the blood from the portal system goes through the anastomosis and it has been considered more likely to remain patent. From

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a clinical point of view, Blalock[3] apparently agrees with the latter opinion, and has stated, "Suture of the divided distal end of the portal vein to the side of the inferior vena cava is preferable to a side-to-side anastomosis, since the opening is more apt to remain patent."

We have reasoned that the side-to-side type may be more likely to thrombose first because blood flows into the anastomosis from two directions. This causes disruption of normal laminar flow and sets up eddy currents, with the result that the central core carrying the platelets is thrown against the walls of the anastomosis, the platelets adhering and catching additional platelets, resulting in occlusion. Second, the pressure drop of blood going through the anastomosis would be less, making less force to disrupt particles adherent to the anastomosis, for it is possible for part of the portal flow to continue to go through the liver.

As there are times when an end-to-side anastomosis cannot be performed because of hemorrhage from varices in the gastroduodenal ligament, it was thought to be desirable to compare the incidence of thrombosis in the two types of anastomosis.

In addition, on the basis of the preceding postulates, the work of Ravdin and Vars,[4] associating anoxemia with liver degeneration, and that of Reynolds[5] associating anoxia with a decrease in liver glycogen, it appeared reasonable that if liver regeneration was proportional to hyperemia, and that if oxygenated blood protected the liver against chloroform poisoning, a direct method of producing hyperemia with oxygenated blood, such as arterialization, by connecting the stump of the portal vein to an artery, might prove of benefit in cirrhosis of the liver. Accordingly, after portacaval anastomosis had been performed, two methods were used. In one a vein graft was sutured to the aorta below the superior mesenteric artery, then to the stump of the portal vein. In the other method, the vein graft was first connected to the right renal artery, then to the stump of the portal vein.

MATERIALS AND METHODS

Lateral or side-to-side anastomosis of the portal vein to the inferior vena cava was performed on anesthetized mongrel dogs through a subcostal incision. Because acute obstruction of the portal vein leads to fatal engorgement of the intestines in dogs,[6*] special clamps, [1, *a* and *b*] previously described by the authors, were used.[7] These clamps allowed blood to continue to flow through both the portal vein and the inferior vena cava while lateral or side-to-side anastomosis was performed. They were sutured in position, thus preventing slipping (Fig. 2, *a* and *b*). The actual technique of using the clamps on dogs was as follows:

After a subcostal incision was made and the intestines were packed off, a Balfour self-retaining retractor was inserted. In order to simulate a partial hepatic block, a ligature was placed above the junction of the superior mesenteric and splenic veins and below the entrance of the pyloric vein. This was

*Made by George P. Pilling and Sons Company, Philadelphia 4, Pa.

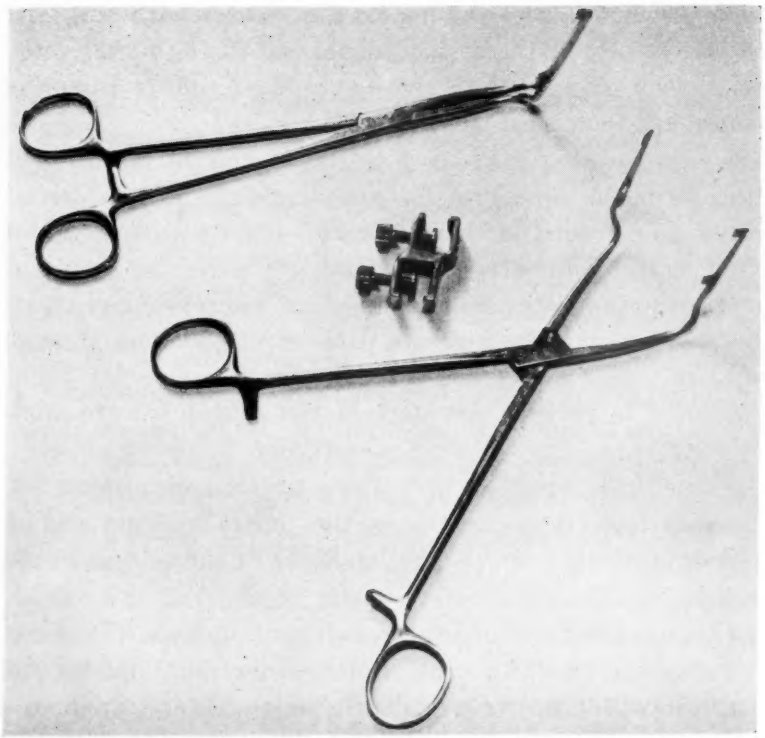

Fig. 1, *a*.—Top, larger clamp for inferior vena cava; center, holding device; bottom, smaller clamp for portal vein.

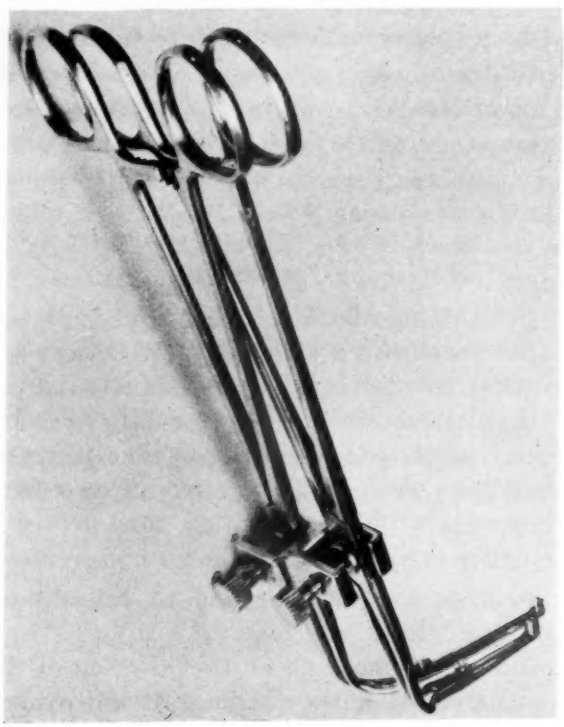

Fig. 1, *b*.—Clamps crossed and maintained in position with holding device.

not tied until after the anastomosis was performed. The peritoneal covering was dissected from the anterior surface of the segment of the portal vein to which the clamp was to be applied. Because of bleeding, it was not desirable to dissect the posterior surface of the portal vein. The previously placed ligature was temporarily occluded until the portal vein was distended with blood. The small clamp was then applied and the vein was maintained in position on the clamp with thumb forceps until the transfixion sutures were placed (Fig. 2, *a*). The large clamp was then applied to the inferior vena cava, without

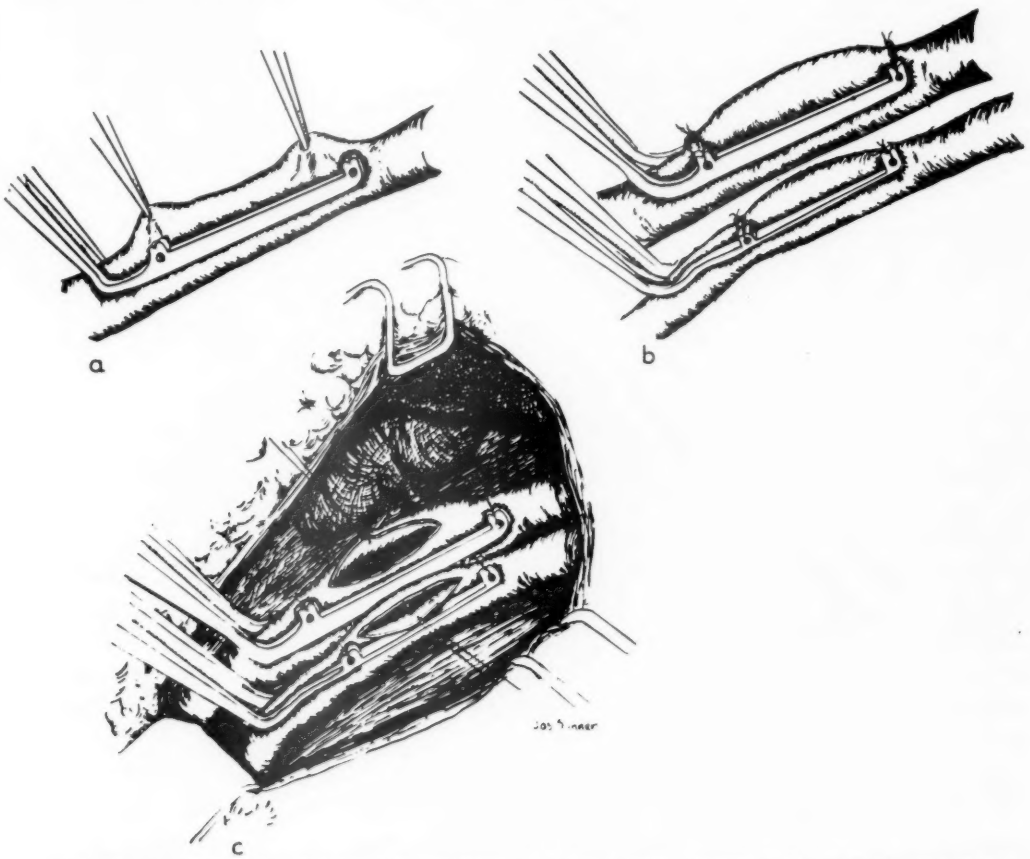

Fig. 2.—*a*, Vein is maintained in position on clamp with thumb forceps until transfixion sutures are placed. *b*, Transfixion sutures are placed and tied. *c*, Segment is excised from inferior vena cava and incision made in the portal vein with traction sutures placed.

dissection of the peritoneum from the anterior surface. The anterior surface was simply lifted with thumb forceps and the large clamp applied directly. A longitudinal slit was made in a closed segment of the portal vein with fine, sharp scissors of the fistula type. It was usually begun by making a tiny transverse incision, then reversing the direction of the blades and making a longitudinal incision (Fig. 2, *c*). A segment was excised from the inferior vena cava using curved tonsil scissors. The intimal surfaces were washed free of blood with saline solution. The posterior suture line was placed in the Blalock manner, that is, the entire posterior suture line was put in with the lips of the two

vessels approximately 1 cm. apart. In this method, it is important that the suture material be braided* rather than twisted silk (Fig. 3, *a*).

An everting running mattress stitch approximating intima-to-intima was used. The suture line was begun at the inferior corner nearest the operator and taken superiorly away from the operator.

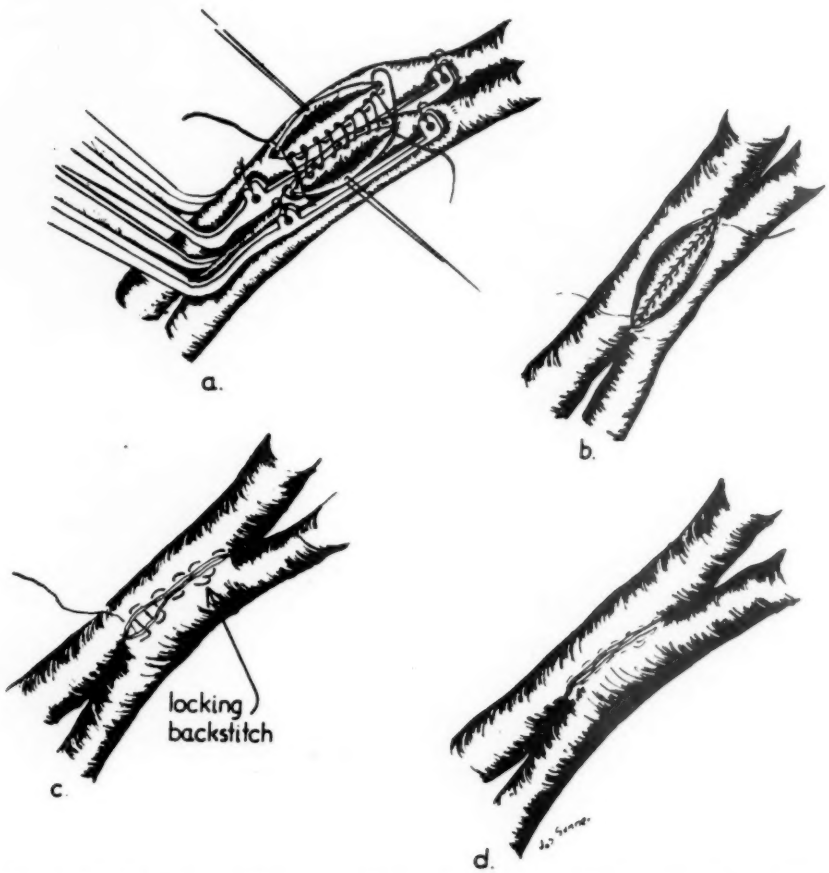

Fig. 3.—Steps showing the method of suture. Everting running mattress suture is used throughout.

After the entire posterior suture line was placed, the two clamps were brought in close approximation by crossing the shank of the smaller in front of the larger, and the loops were tightened by lifting with small thumb forceps until the posterior lips of the two vessels were closely approximated (Fig. 3, *b*). The everting mattress suture was continued around the superior corner and approximation of the anterior lips of the two vessels begun (Fig. 3, *c*). Two or three back stitches were then taken to keep the suture line taut. The anterior suture line was then placed and secured by tying to the original end at the lower corner (Fig. 3, *d*). According to present concepts of suturing, it would be more orthodox to break the suture line by tying to interrupted sutures at three points; however, the preceding technique works and is faster. The clamp

*Deknatel 0000 braided silk on a No. 94 needle was used throughout.

on the inferior vena cava was released first because of low pressure on this side of the shunt. Any bleeding points were controlled with interrupted mattress sutures. The portal clamp was slowly released and removed and the previously placed ligature on the portal vein superior to the anastomosis was tied.

An end-to-side anastomosis was performed in similar fashion, but differed in the following points. Speed is an important factor as the portal vein was completely occluded, and in fifteen to twenty minutes the intestines would become fatally engorged.[6] Therefore, all steps that could be, were carried out before the portal vein was divided.

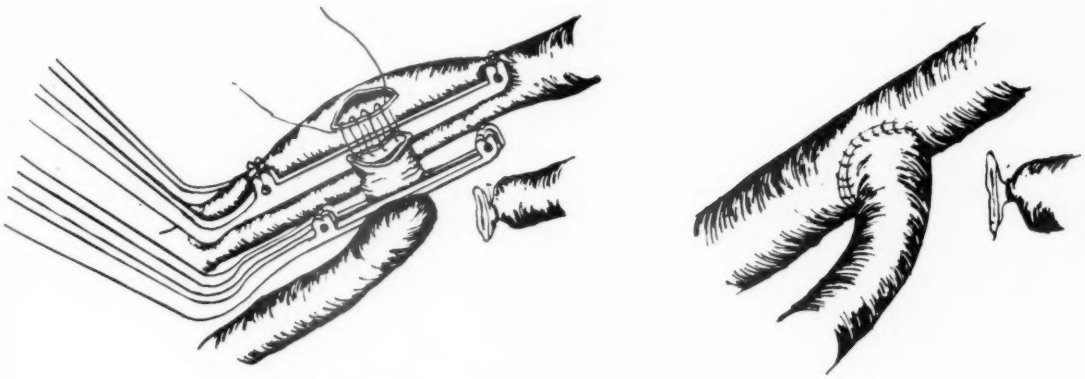

Fig. 4.—End-to-side anastomosis. Continuous running Carrel stitch is used throughout. Note ligated portal stump.

First the portal vein had to be dissected free in its entire circumference. The larger clamp was placed on the inferior vena cava and transfixion sutures were inserted. A segment was then excised from its anterior surface. The ligature on the portal vein was tied by an assistant as the operator applied the smaller clamp and divided the vessel. The smaller clamp was applied in such a fashion that the end of the portal vein became anterior. A single transfixion suture could have been applied through the hole at either end of the clamp. The two clamps were manipulated to hold the sides of the two veins in close approximation and were locked in position by the holding clamp. This enabled the assistant to steady both clamps with one hand. Because speed was a factor, a running Carrel stitch was used. It was begun at the lower corner close to the operator, and was taken away (Fig. 4, *a*). The portal vein was stretched to the size of the segment excised from the inferior vena cava. Back stitches were taken at the superior corner and the original suture line was tied to its end at the lower corner (Fig. 4, *b*).

Venous pressures in certain experiments were taken by inserting a short arterial catheter in the superior mesenteric vein through a tiny purse string suture of 00000 Deknatel. This was connected to a saline manometer held in constant position at the level of the heart.

Arterialization of the liver from the aorta was carried out as follows:

1. The approach was through a thoracoabdominal incision of the type described by Satinsky[8] for portacaval anastomosis.

2. Side-to-side anastomosis of the portal vein to the inferior vena cava was first performed. The same ligature described previously was placed around the portal vein and tied. The pyloric vein was divided and ligated. The portal vein clamp was used to clamp the upper end of the portal vein in a similar fashion as described for end-to-side anastomosis. The vein was then divided. The vein graft held by two curved hemostats in a special holding clamp previously described* (Fig. 5) was sutured to the stump of the portal vein.[8] The posterior suture line was inserted in the same fashion as described for the side-to-side anastomosis. It was anchored in position at each end by an interrupted suture. The anterior row was approximated with interrupted mattress sutures. Anastomosis to the aorta was carried out using a clamp similar to the

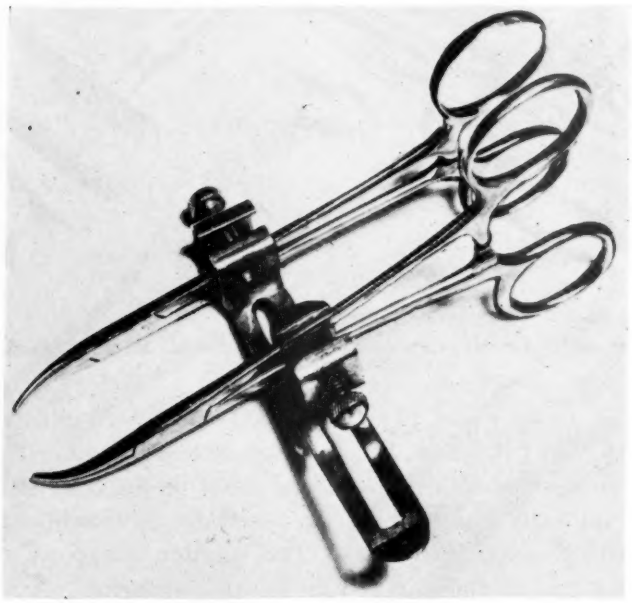

Fig. 5.—Holding clamp with two hemostats locked in position.

portal vein clamp, but of heavier design and with serrations on the blades. This was placed on the aorta below the superior mesenteric artery and the previously described transfixion sutures were inserted. The inferior end of the vein graft was sutured to the aorta by one of two methods, either by using the holding clamp as shown in Fig. 6, or by using a second method similar to that used in performing an end-to-side portacaval anastomosis. In the latter method, the inferior end of the vein graft was clamped by the portal vein clamp in a manner to avoid twisting, and, with its end turned up, was approximated to the aorta on the left side, in the same relative position as that used in end-to-side portacaval anastomosis. The two clamps were then locked in position using their specific holding device and a continuous everting mattress suture was inserted as described for the lateral anastomosis. However, here the suture line was tied to an interrupted suture at each corner and on the right lip of the anas-

*Made by George P. Pilling and Sons Company, Philadelphia 4, Pa.

tomosis. The clamp on the portal vein was released first. Bleeding points were controlled with interrupted sutures before releasing the clamp on the aorta. Suture of the vein graft to the right renal artery was performed in a similar fashion except that here single interrupted sutures were used throughout, because of the small size of the artery.

DISCUSSION

Side-to-side anastomosis was performed forty times (Table I). One animal died of aspiration vomitus within twenty-four hours, one was sacrificed within one week, and one died of intussusception three days postoperatively. The remaining thirty-seven animals were viable two weeks or a greater period of time.

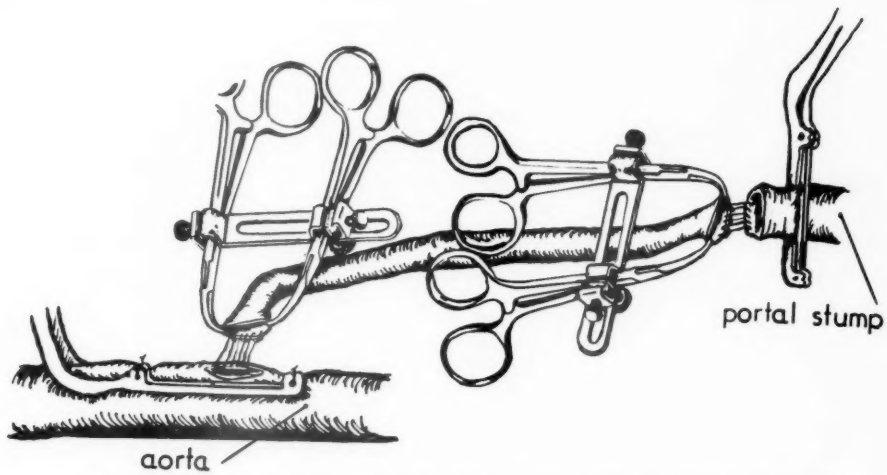

Fig. 6.—Method of suture of vein graft from aorta to portal stump.

Eleven of the anastomoses had an opening of 1.2 cm. or less. In nine of the eleven no segment was cut from the inferior vena cava. Three of the nine animals were completely thrombosed, giving a percentage of 33 per cent. In addition, two were partially thrombosed with the lumen occluded 50 per cent or more. The over-all per cent of thrombosis in this group, with the length of the stoma 1.2 cm. or less, was 27 per cent. In the remaining twenty-six anastomoses the stoma was 1.5 to 2 cm. in length. There were no thromboses in this group, even though in six animals no segment was cut from the inferior vena cava. Post-mortem examination revealed that, with two exceptions, all of the anastomoses 1.8 to 2 cm. at the time of operation were 1.3 cm. or greater in length at the end of two weeks or a longer period of time.

End-to-side anastomosis was performed twenty-six times (Table II). Tying off the pyloric vein did not give significant difference in results in the animals in which it was performed. In all of the animals a segment was cut from the inferior vena cava. Eight animals died of postoperative complications from twenty-four hours to five days later. One died of shock, one of generalized hemorrhage, one of venous obstruction from angulation of the portal vein, another of peritonitis from a sponge inadvertently left in, one died of pneu-

TABLE I. SIDE-TO-SIDE ANASTOMOSIS, PORTAL VEIN TO INFERIOR VENA CAVA

WEIGHT (POUNDS)	LENGTH OF ANASTOMOSIS (CM.)	SEGMENT* VENA CAVA	FATE
37	.8	N. C.	Patent, sacrificed 9 wk. later
40	.8	N. C.	Thrombosed, sacrificed 7 wk.
54	.8	N. C.	Patent, partially thrombosed, sacrificed 2 wk.
35	1.5	N. C.	Patent, sacrificed 4 wk.
28	1.5	N. C.	Patent, sacrificed 4 wk.
15	1.5	N. C.	Patent, sacrificed 1 wk.
40	1.5	N. C.	Patent, sacrificed 5 wk.
40	1.2	N. C.	Patent, partially thrombosed, ⅓ lumen open, sacrificed 2 wk.
40	1.6	N. C.	Patent, sacrificed 2 wk.
35	1.2	N. C.	Patent, sacrificed 6 wk.
40	1.2	N. C.	Patent, died aspiration vomitus in 24 hr.
34	2.1	N. C.	Patent, sacrificed 3 wk.
40	1.2	N. C.	Thrombosed, sacrificed 2 wk.
28	1.2	N. C.	Patent, sacrificed 2 wk.
27	1.2	N. C.	Thrombosed, sacrificed 2 wk.
28	1.2	S. C.	Patent, sacrificed 2 wk.
47	1.2	S. C.	Patent, sacrificed 3 wk.
43	2	S. C.	Patent, 4 wk., death from pneumonia
47	2	S. C.	Patent, 3 wk., death from pneumonia
30	1.5	S. C.	Patent, sacrificed 2 wk.
46	2	S. C.	Patent, sacrificed 2 wk.
35	2	N. C.	Patent, sacrificed 3 wk.
35	2		Patent, death from intussusception 3 days
32	1.8	S. C.	Patent, 0.8 cm. long, sacrificed 14 days
32	1.8	S. C.	Patent, 0.8 cm., died from distemper 18 days
33	1.9	S. C.	Patent, 1.2 cm., sacrificed 14 days
31	1.9	S. C.	Patent, 1.3 cm., died of pneumonia 4 wk.
24	1.9	S. C.	Patent, 1.4 cm., sacrificed 14 days
40	2	S. C.	Patent, 1.3 cm., sacrificed 14 days
30	1.9	S. C.	Patent, 1.3 cm., sacrificed 7 days, distemper
29	1.8	S. C.	Patent, 1.3 cm., sacrificed 14 days
30	1.8	S. C.	Patent, 1.3 cm., died 3 wk., distemper anastomosis
32	1.8	S. C.	Patent, 1.1 cm., sacrificed 4 wk.
35	1.8	S. C.	Patent, 1.1 cm., died of distemper 4 wk.
36	1.8	S. C.	Patent, 1.3 cm., sacrificed 3½ mo.
33	1.8	S. C.	Patent, 1.1 cm., died distemper 3 wk.
33	1.8	S. C.	Patent, 1.3 cm., sacrificed 2 wk.
32	1.8	S. C.	Patent, 1.3 cm., sacrificed 2 wk.
33	1.8	S. C.	Patent, 1.4 cm., sacrificed 2½ mo.
32	1.8	S. C.	Patent, 1.3 cm., sacrificed 2 wk.

*S.C. Segment excised from inferior vena cava.
N.C. Incision made in inferior vena cava, but no segment excised.

monia in two days, and in three cases the cause of death was not determined. In the remaining seventeen animals, four anastomoses were completely thrombosed (24 per cent) and five were partially thrombosed, presenting a lumen of 0.5 cm. or less. In this type of anastomosis the length of the incision in the inferior vena cava was made 2 to 3 mm. larger than the portal vein when it was fully distended with blood. In the average dog of 30 pounds this would approximate 1.2 cm. After three weeks or more this tends to contract to 0.6 to 0.7 cm.

In the side-to-side anastomosis the average length of the incision was 1.9 cm. This tends to narrow to approximately 1.2 cm. Cutting a segment from the inferior vena cava in the preceding series apparently did not overcome the increased incidence of thrombosis in an end-to-side anastomosis performed with

TABLE II. END-TO-SIDE ANASTOMOSIS, PORTAL VEIN TO INFERIOR VENA CAVA

WEIGHT (POUNDS)	TIME (MIN.)	PYLORIC VEIN	SEGMENT VENA CAVA	FATE
30	13	Not tied off	S. C.*	Thrombosed, sacrificed 3 wk. later
31	10	Not tied off	S. C.	Patent, sacrificed 3 wk.
25	8	Tied off, not sectioned	S. C.	Patent, died 48 hr.
25	10	Tied off, not sectioned	S. C.	Patent, sacrificed 9 days
25	14	Not tied off	S. C.	Patent, died 72 hr., cause unknown
25	13	Tied off, sectioned	S. C.	Patent, died of shock, 5 hr. postop.
29	8	Tied off, sectioned	S. C.	Patent, died of pneumonia 2 days
27	8	Tied off, sectioned	S. C.	Thrombosed, sacrificed 10 wk.
31	13	Tied off, sectioned	S. C.	Partially thrombosed opening 2 mm. diam.; sacrificed 2 wk.
29	12	Tied off, sectioned	S. C.	Patent, died 16 days, anaerobic infection
30	12	Tied off, sectioned	S. C.	Patent, sacrificed 3 mo., completely thrombosed
33	12	Not tied off	S. C.	Patent, sacrificed 24 days, partially closed, 0.6 cm. long
35	14	Not tied off	S. C.	Partially closed, 0.3 cm., sacrificed 24 days
34	12	Not tied off	S. C.	Partially closed 0.5 cm., sacrificed 16 days
33	12	Not tied off	S. C.	Patent, anastomosis, died 4 days, of peritonitis, sponge left in
34	18	Not tied off	S. C.	Partially thrombosed, 0.6 cm., sacrificed 2 wk.
32	18	Not tied off	S. C.	Patent, died of generalized hemorrhage 4 days
31	15	Not tied off	S. C.	Patent, died 5 days, cause of death unknown
50	16	Not tied off	S. C.	Patent, died of portal obstruction from twisted vein in 24 hr.
30	15	Not tied off	S. C.	Patent, 0.6 cm., sacrificed 2 wk.
30	15	Not tied off	S. C.	Patent, 0.3 cm., sacrificed 3 wk.
30	16	Not tied off	S. C.	Patent, 0.6 cm., sacrificed 2 wk.
32	14	Not tied off	S. C.	Patent, thrombosed, 0.3 cm., sacrificed 3 wk.
30	19	Not tied off	S. C.	Patent, 0.6 cm., sacrificed 2 wk.
35	19	Not tied off	S. C.	Patent, 0.6 cm., sacrificed 2 mo.
33	20	Not tied off	S. C.	Completely thrombosed, sacrificed 2 wk.

*S.C. Segment excised from inferior vena cava.
N.C. Incision made in inferior vena cava, but no segment excised.

a continuous Carrel stitch. The Carrel stitch did not give as smooth an anastomosis as the everting mattress. Minute thrombi tended to form along the suture line.

Experimentally these did not give trouble, but clinically they have been observed occasionally to become a site of a bacterial arteritis similar to bacterial endocarditis, with minute thrombi breaking off, giving rise to infarction in various parts of the body. The question of the quantity of blood flowing through the liver after an adequate side-to-side portacaval anastomosis may be discussed at this point. In a dog anesthetized with Nembutal (1 gr. per 5 pounds of body weight), a normal portal vein pressure (in the segment superior to the pyloric vein) may be taken at 5 to 6 cm. of water. If the portal vein is occluded with a temporary ligature between the pyloric vein and the superior mesenteric-splenic confluence, the pressure in the section above the ligature tends to fall 1 to 2 cm. of water and below the ligature tends to make an acute rise to 40 to 60 cm. of water. If the ligature is released and a side-to-side portacaval anastomosis is performed, the pressure in the portal vein tends to fall to 3 to 4

cm. of water. If the same ligature previously placed is again tightened, the pressure in the segment above the ligature rises 1 to 2 cm. of water. This indicates that blood is flowing mainly in the direction of the anastomosis and is entering the anastomosis from two directions, substantiating our earlier view. If, after the ligature of the portal vein is tied, the only large tributary (pyloric vein) entering the section superior to the ligature is also occluded, the pressure in this section tends to fall 1 to 1½ cm. of water. This indicates that the quantity of blood flowing through an end-to-side portacaval anastomosis with the pyloric vein tied is greater than that going through a side-to-side anastomosis with the pyloric vein open and a ligature placed as described above. Giving the end-to-side anastomosis a slight increase in rate of flow was one of the conditions of the experiment. The rise in portal vein pressure, with the special portacaval anastomosis clamps in place, was not great. With the portal vein clamp in place, the venous pressure rose to 9 to 12 cm. of saline solution, depending on the segment enclosed by the clamp. This is quite different from a rise of 40 to 60 cm. of water experienced when complete occlusion of the portal vein takes place.

From the above information it may be reasoned that in a patient with cirrhosis having a pressure of 30 cm. of water, very little or no blood would flow through the liver after an adequate side-to-side anastomosis. If in a normal liver the pressure of 7 to 8 cm. of water, in the upper end of a portal vein, falls after a side-to-side portacaval anastomosis and rises if it is separated from the anastomosis, it would appear that little or no blood would appear to go through a liver requiring a pressure of 30 cm. of water.

Arterialization of the liver by graft between the aorta and the stump of the portal vein has been performed four times. The grafts have functioned well from periods of three weeks to four months, without thrombosis. A pressure of 38 cm. of water was noted in one graft after two months. On sacrifice of this dog three months after operation, hypertrophy of the right auricle and dilatation of the pulmonary artery was noted. In three animals in which the graft was connected to the right renal artery, thrombosis occurred in two to four days. The third graft of this type was patent two and one-half months later, when the animal was sacrificed.

Studies are in progress to determine specific effects on liver function. These will be reported at a later date. Suffice it to say, however, that, microscopically, an absence of lipoid has been reported. This appears promising, as recently a group of writers have reported that portal obstruction is proportional to deposition of lipoid rather than to fibrosis.[10]

CONCLUSION

In a consecutive series of twenty-seven dogs, portacaval anastomosis has been performed without thrombosis. The length of the stoma in this series was 1.5 to 2 cm. and an everting continuous mattress stitch was used. Special clamps previously described by the authors were used. When the stoma was made 1.2 cm. or less, the incidence of thrombosis rose to 27 per cent. In a series of twenty-six animals with end-to-side anastomosis employing a running Carrel

stitch, the incidence of thrombosis was 25 per cent. Two methods of arterialization of the liver are described. A vein graft from the stump of the portal vein to the aorta after portacaval anastomosis appears most satisfactory in so far as the factor of thrombosis is concerned. Further studies are in progress.

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THE COEXISTENCE OF GOITER AND DEAF-MUTISM

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IN THE course of the last twenty years or so it has been becoming increasingly more obvious that, just as diabetes is not a primary disease of the pancreas, goiter is not merely a primary affection of the thyroid gland. The role of hypophyseal hyper- or hypoactivity has come to the foreground so that a number of symptoms formerly ascribed to thyroid malfunction we now know to be of pituitary origin. There remain, however, a number of symptoms associated with evidence of thyroid disease whose mechanism of development is unknown. One of these is deaf-mutism, which, although not commonly present, does show a definitely higher incidence in goiter patients than in the population in general.

As far as could be ascertained, the first mention of the coexistence of goiter with deaf-mutism is found in an article by Bircher[1] in 1883. Since then a small number of publications have confirmed his findings. At present we wish to review the scant literature and add a few cases of our own.

Bircher noted that the incidence of deaf-mutism in the general population of countries with a low goiter rate was around 3 per 10,000, while in Switzerland, a country with a notoriously large number of goiter patients, the incidence ranged between 24 and 250 per 10,000.

In Switzerland again, the frequency with which deaf-mutism occurred in various parts of the country varied directly with the incidence of endemic goiter.

In 1934 Stott and Gupta[4] reported on the incidence of endemic goiter and cretinism in certain Indian provinces. They found that in the endemic goiter areas the incidence of deaf-mutism was 23 per 10,000. Of these deaf-mutes, 90 per cent also had an enlarged thyroid gland.

McCarrison, in his article[2] stated that "87 per cent of all cretins are deaf-mutes to a greater or lesser degree."

In our small series of eighty-four patients admitted to this hospital with a diagnosis of nontoxic goiter over a period of five years, four suffered of varying degrees of deaf-mutism. This means that 4.8 per cent of our goiter patients were also deaf-mutes. If we accept Bircher's figures on the incidence of deaf-mutism in a nongoiterous population we find that in our series the incidence of deaf-mutism was 160 times as high as it would be expected to be in a similar group with no thyroid enlargement. None of our patients showed evidence of cretinism or myxedema. Similarly, Brain[5] failed to find cretinism in his cases of associated goiter and deaf-mutism.

There are thus apparently two different groups of patients: one in which goiter and cretinism are associated with deaf-mutism and another, apparently much less numerous group, in which the goiter and deaf-mutism are not associated with any mental changes or low basal metabolism rate.

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The sex incidence in Brain's cases was about even; in our group three of the patients were female and one was male.

The geographic distribution of the cases apparently follows closely that of endemic goiter, that is, the incidence is highest in areas poor in iodine.

The age incidence varies in different reports. In those areas where the deaf-mutism was associated with cretinism both conditions existed since the earliest childhood. In Brain's cases the deaf-mutism was congenital while the goiter did not appear until the second or later first decade of life. In three of our cases the swelling in the anterior neck had been present since early childhood and in the fourth it appeared at the age of 18 years.

A number of theories have been advanced to explain the coexistence of goiter and deaf-mutism. Professor Nager of Zürich (cited by Stott and Gupta) explained the cretinous deaf-mutism by a myxomatous thickening of the submucous tissues of the middle ear. He has demonstrated these changes on microscopic sections and believed them to occur at about the sixth intrauterine month. McCarrison, speaking of the same type of patient, believed that the defect may be caused in part by the infiltrated tongue, aural mucosa, eustachian tubes, and nasopharynx, but that it is mainly dependent on imperfect development, as well as infiltration of the higher brain centers, and on the lack of receptivity of the nerves.

However, Brain's and our cases, as was already mentioned, were not myxedematous. Brain therefore believed that the association of deaf-mutism with goiter may be regarded as an example of linkage in human inheritance. In his group only those children that inherited the blood group of the goiterous parent also inherited the goiter.

In only two of our patients could we elicit a history of goiter in the family. In one, two distant paternal relatives were said to have had enlarged thyroid glands; in the other a paternal and a maternal aunt were known to have goiters.

Murray and Wilson[6] believed with Brain that what is inherited is not goiter, cretinism, or deaf-mutism as such, but rather some defect in iodine utilization.

In none of the published reports is the exact pathology of the thyroid gland mentioned. However, all authors seem to refer to diffuse colloid goiter with or without hypofunction. All of our patients underwent thyroidectomy, and microscopic examination revealed the presence of one or multiple adenomas in three cases, two of which were associated with a nodular colloid goiter. The fourth patient was reported as having a simple nodular colloid goiter.

The picture throughout is thus characteristic of iodine deficiency goiter. Yet all the patients live in or in the vicinity of Beirut, a town on the seacoast where there is presumably an adequate supply of iodine in the food and water. This seems to bear out Brain's view that the underlying mechanism of the condition is an abnormally high iodine requirement.

The treatment for these cases is both medical and surgical. As in all other adolescent goiters, iodine therapy should be given a trial first and surgery

resorted to if that fails. The relatively high incidence of carcinoma in the so-called nodular goiter of young patients makes surgery imperative in any case that does not respond to iodine therapy.

As regards the deaf-mutism, much can be done for these patients if their mental level is normal. There is one center in this town directed by Swiss specialists that teaches these patients to speak and lip-read and two of our patients have been referred to them.

CASE REPORTS

M. W. S. (No. 53371), a 20-year-old unmarried woman, was admitted on Jan. 16, 1949, with a diffuse swelling in the anterior neck of two years' duration. Family history failed to elicit the presence of goiter in any members and the past history was negative except for a tonsillectomy at the age of 17 years.

On admission there were no signs of toxicity. Basal metabolism rate was +8 per cent. Physical examination revealed that in addition to a diffuse, moderately pronounced swelling in the anterior neck the patient was hard of hearing and spoke with a pronounced lisp. A partial thyroidectomy was performed and she made an uneventful recovery.

Pathologic examination of the specimen revealed a nodular colloid goiter.

N. A. N. (No. 65096), a 13-year-old deaf-mute boy, had complained of masses in the anterior neck since the age of 7 years. Intake of iodine resulted in a temporary regression of the lesions, but for a short period prior to admission the patient had complained of dyspnea on exertion. The family history revealed that three siblings and both parents were normal. However, two distant paternal relatives had goiter and his greatgranduncle was reported to have been a deaf-mute. Physical examination revealed a large nodular goiter. Laryngoscopy and otoscopy were negative. A roentgenogram revealed a small retrosternal extension of the goiter. The basal metabolism rate preoperatively was −22 per cent. A subtotal thyroidectomy was performed. Following operation the patient developed a positive Chvostek sign and the serum calcium dropped from 11.2 mg. per cent preoperatively to 7.2 mg. per cent. The postoperative course was otherwise uneventful and the incipient tetany responded to medical therapy. This patient again seemed of about normal intelligence.

Pathologic report showed multiple adenomas of the thyroid gland.

S. K. H. (No. 64555), a 19-year-old Bedouin girl, came in with a nodular mass in the anterior neck. She was a partial deaf-mute, so that the history had to be taken from her relatives. The swelling was apparently noted in early childhood and had been slowly increasing in size ever since. There were signs of mild tracheal compression by x-ray examination and the patient complained of dyspnea and palpitations. An electrocardiogram revealed a bundle of Kent syndrome. The basal metabolism rate was +11 per cent.

Physical examination was negative except for a nodular goiter and a soft apical systolic murmur. Laryngoscopy was negative. A subtotal thyroidectomy was performed. Histologically the lesion was found to consist of multiple adenomas in a nodular colloid goiter.

H. N. A. (No. 66323), a young deaf-mute Iraqi woman, had a chief complaint of an unsightly mass in the anterior neck, which had been present ever since she could remember. Her paternal and maternal aunts were both said to have goiters, but there was no deaf-mutism in any other member of the family. The patient also complained of exertional dyspnea and palpitations. Basal metabolism rate was +7 per cent. Physical examination revealed an intelligent young person with a moderately large, diffuse goiter and a 4 by 4 cm. nodule in the region of the isthmus. Subtotal thyroidectomy was performed. The pathologist's report was fetal adenoma of the thyroid gland and nodular colloid goiter.

SUMMARY

The literature on the coincidence of goiter and deaf-mutism has been briefly reviewed and four cases have been discussed.

ADDENDUM

Since this article went to print we have had one more case of deaf-mutism associated with goiter. This brings the total number of cases to five in the course of five years.

The patient was a 14-year-old boy of normal intelligence with a large adenoma involving the isthmus and right lobe of the thyroid. Basal metabolism rate and laryngoscopy were within normal limits. Family history revealed the presence of a nontoxic goiter in the patient's mother, but was otherwise negative.

In spite of the patient's deaf-mutism the usual precautions as regards the recurrent laryngeal nerve were taken since, as we already mentioned, these patients can be taught to speak.

My sincerest thanks are due to Dr. J. J. McDonald and Dr. Y. Jidejian who have allowed me to use their case material. I am also particularly indebted to Dr. J. J. McDonald for having kindly consented to read and correct the manuscript.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS

Report of Two Patients Treated Surgically With Survival

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RUPTURE of a previously normal esophagus can occur from violent muscular action without the intervention of trauma by instrumentation, swallowed foreign body, or external violence. Since spontaneous esophageal rupture is rare and since it may mimic an intra-abdominal or some other intrathoracic catastrophe, it is frequently not considered among diagnostic possibilities. This is particularly serious because ordinary surgical principles, together with the results of previously reported cases, indicate the wisdom of immediate repair following prompt diagnosis. It seems important, therefore, to present two cases with recovery in order to call attention again to this unusual lesion.

Case 1.—After negative exploration for suspected perforated peptic ulcer, hydropneumothorax appeared. At thoracotomy, esophgeal rupture was verified and repaired. Recovery occurred after dehiscence of the thoracic wound and drainage of empyema.

A 55-year-old Negro brakeman was admitted because of projectile vomiting of brown fluid for four hours and severe continuous pain in the left lateral trunk from flank to axilla. History was not obtainable as to the onset of illness in relation to food ingestion or if pain preceded or followed vomiting. The patient had noted rigidity of the abdomen but no abdominal pain.

Examination revealed an acutely ill Negro man lying with the knees flexed on the abdomen in extreme discomfort. Temperature was 101.2° F. rectally. Blood pressure was 120/80, pulse 96, and respiration 30. The mucous membranes and nail beds were pale. Respiration was shallow, with splinting of the thoracic wall. Chest examination revealed hyper-resonance and distant breath sounds on the left. Abdominal examination revealed normal abdominal respiration and boardlike rigidity, but no tenderness. A Levine tube was passed, and thin, black, odorless material was aspirated.

Laboratory studies revealed the following: hemoglobin 13 Gm., red cell count 5 million, white cell count 9,100, and amylase 17 units.[1] Urinalysis was negative except for 10 to 15 white cells per high power field. A tentative diagnosis of ruptured intra-abdominal viscus was made. Exploratory laparotomy under general anesthesia, including visualization of the pancreas, revealed no abnormality. Seven hours postoperatively the blood pressure dropped to 70/40 and the pulse was 144. Electrocardiogram showed isoelectric T wave in Lead I and slurring of the QRS complex. The patient responded to 500 c.c. of whole blood, oxygen, and digitoxin, but continued to vomit bloody material. He again lapsed into shock with a systolic blood pressure of 50. Physical examination and fluoroscopy of the chest indicated fluid and air in the left pleural space. Accordingly, thoracentesis was performed and 450 c.c. of benzidine-positive, alkaline, brownish fluid were obtained. A roentgenogram of the chest after the administration of Lipiodol through the Levine tube revealed leakage from the esophagus into the left pleural space just above the diaphragm.

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Twelve hours after admission, exploratory thoracotomy under endotracheal anesthesia through the left eighth rib bed revealed a large amount of brownish-red fluid and old food particles. Two cm. above the diaphragm there was a 3 cm. longitudinal rent through the entire thickness of the esophagus. A smaller rent through the mucosa only was noted on the posterior wall. There was no ulcer of the esophagus or other lesion to predispose to the rupture. A careful search revealed no foreign body. The rent in the esophagus was closed with fine silk in two layers. The pleural cavity was carefully irrigated with saline solution and the incision closed in layers with a catheter in the pleural space for aspiration of air and fluid and the instillation of streptomycin and penicillin. During the procedure it was difficult to maintain the patient's blood pressure with whole blood and intravenous fluids and he was returned to the ward in poor condition.

Postoperatively he received penicillin and streptomycin intramuscularly and intrapleurally. Daily aspiration of the pleural cavity was carried out; by the fourth postoperative day only 10 c.c. of fluid were obtained. On the ninth postoperative day the thoracotomy wound separated, with evacuation of 100 c.c. of foul, reddish-brown fluid. Left thoracentesis obtained 550 c.c. of foul, brown material. Chest roentgenogram revealed the left lung field to be obscured by a dense homogenous shadow which was particularly dense in the medial two-thirds of the lung field posteriorly. On the tenth postoperative day, open drainage with resection of the seventh left rib was performed under local anesthesia. A large posterior empyema pocket which did not communicate with the esophagus was encountered and drained. The empyema space gradually obliterated and the patient was in excellent condition on the twentieth day after drainage.

In retrospect, this case presented on admission symptoms and signs which should have led to x-ray examination of the chest. If this had been obtained, delay in diagnosis and needless laparotomy would have been avoided, for the correct diagnosis would have been obvious.

CASE 2.—While under observation for suspected perforated peptic ulcer this patient developed pyopneumothorax. Rupture of the esophagus was demonstrated by barium swallow. Recovery occurred after unsuccessful closure of the esophagus and drainage of the periesophageal abscess.

A 66-year-old white woman was admitted with the chief complaint of severe, cramping, mid-epigastric pain of 7 hours' duration. The pain began suddenly while the patient was eating, bored through to the back, radiated to the shoulder, and was aggravated by breathing. She broke out in a sweat and became ashen.

Physical examination revealed an acutely ill woman with a systolic blood pressure of 70, temperature of 100.4° F. pulse of 120, and respiration of 30. The skin was slightly cyanotic. Chest examination revealed fine râles in both lungs posteriorly. There was abdominal respiration, no abdominal tenderness, and no rebound tenderness or spasm. The abdomen was tense throughout, and peristaltic sounds were normal. Cardiac examination was negative.

Laboratory work revealed the following: white count of 13,500 with 73 per cent segmented polymorphonuclear cells, 16 per cent bands and 3 per cent juveniles; red cell count of 3.9 million; hemoglobin 13 Gm. per 100 c.c.; hematocrit 38; serum amylase 137.[1] Urinalysis showed 1+ albumin, 1+ sugar, and 1 to 5 white cells per high power field. The electrocardiogram revealed a slight ST depression, a diphasic T wave and slight T wave inversion in V_L, with a rate of 125.

The impression on admission was ruptured ulcer without general peritonitis. Pancreatitis, myocardial infarction, mesenteric thrombosis, and dissecting aneurysm were considered also. Because of the patient's condition and evidence that she was caring for the possible peritoneal insult adequately, observation was advised.

She was given whole blood (500 c.c.), nasal oxygen, penicillin, and streptomycin. Ten hours after admission she complained of epigastric pain radiating through to the back and to the neck. She vomited once. Gastric lavage revealed only blood-tinged mucus. A roent-

genogram taken on the third hospital day showed a left basilar pneumonia and pleurisy and on the fifth day revealed fluid pocketed off in the left chest and air either above or below the diaphragm. Thoracentesis the following day yielded 35 c.c. of straw-colored fluid, which was negative for gastric secretions. At this time the possibility of rupture of the esophagus was first considered. Her clinical course was satisfactory until the tenth hospital day, when she developed dyspnea followed by sharp pain in the left lower chest and a rise in temperature. On the seventeenth day, a roentgenogram revealed barium escaping from the left anterior surface of the esophagus just above the cardia. On the twentieth day, left thoracotomy revealed the pleural cavity filled with fibrin and rupture of the lower third of the esophagus which communicated with the fibrinous empyema. Closure of the rupture of the esophagus, decortication, and closed drainage of the empyema were carried out.

The patient had a stormy postoperative course characterized by sepsis. She was fed by tube for one week and then started on liquids. On the eleventh postoperative day, a left eighth rib thoracotomy was performed with drainage of a peri-esophageal abscess about the area of esophageal repair. The patient continued to run a septic course for about seven days and then gradually improved. She passed food particles through the thoracotomy tube for eight days. Twenty days later all drainage had ceased.

A gastrointestinal x-ray study on the thirty-sixth postoperative day showed only evidence of a hiatus hernia with a short esophagus.

On admission, though there were symptoms suggestive of chest disease, the patient did not present physical signs suggestive of thoracic disease. A roentgenogram of the chest was obtained 3 days after admission because of subsequent development of chest signs. Had a chest roentgenogram been obtained immediately because of the symptoms, an early diagnosis might have resulted.

DISCUSSION

Spontaneous rupture of the esophagus, a relatively rare lesion, although first recognized pathologically by Boerhaave in 1724,[1] has been treated successfully only since the advent of modern thoracic surgery and the use of antibiotics. The first report of a survival after definitive surgical treatment is that of Barrett in 1947.[2] The disease is characterized pathologically by a longitudinal tear of the esophagus just above the diaphragm and differs from traumatic and other types of perforation of the esophagus in that there exists no previous demonstrable lesion of the esophagus which predisposes to its rupture. The tear allows the escape of esophageal and gastric contents into the mediastinum and one or both pleural cavities, more frequently the left. Although in both of the cases reported here a gastric tube was passed before the correct diagnosis was established from the clinical history and course, it was felt that the rupture of the esophagus was spontaneous and occurred before gastric lavage.

The picture usually presented immediately after this accident suggests an upper abdominal catastrophe, but shortly symptoms and signs develop in the thorax. The diagnosis, except in a few cases in which there is subcutaneous emphyema, must be made by keeping the possibility of this lesion in mind in cases of acute severe pain in the upper abdomen and lower chest. There are several factors in the history and physical findings which should suggest the possibility of esophageal rupture and lead to an early diagnosis in the majority of cases. There is usually a history of severe vomiting, often

TABLE I

AUTHOR	PERFORATION	DURATION	OPERATION	COMPLICATIONS
1, 2. Graham (1944)[4]	(1) No data	No data	Drainage of empyema	No data
	(2) No data	No data	Drainage of empyema	No data
3. Cummings (1946)[5]	(3) No data	No data	No data	Bilateral hydropneumothorax; food in pleural cavities
4. Barrett (1947)[3]	(4) Small perforation in the posterior wall about 1½" above the diaphragm	10 hr.	1. Purse-string closure with catheter in pleural space 2. Drainage of mediastinal abscess, enterostomy	Leak of esophagus; mediastinal abscess Lung abscess
5. Frink (1947)[6]	(5) Not explored	5 days	1. Closed intercostal drainage 2. Open drainage of empyema	Left empyema
6. Olsen and Clagett (1947)[7]	(6) Vertical tear 3 to 4 cm. just above diaphragm	3 hr.	Repair in 3 layers	None
7. Moore and Murphy (1948)[8]	(7) 4 cm. rent in anterolateral esophagus just above diaphragm	16 days	1. Open drainage of empyema 2. Jejunostomy 3. Esophagogastroplasty	Left empyema
8, 9, 10. Lynch (1949)[9]	(8) Linear laceration in bottom of empyema cavity	60 hr.	1. Intercostal drainage 2. Open drainage of empyema mediastinal abscess; jejunostomy 3. Four unsuccessful attempts at repair	Left empyema; mediastinal abscess
	(9) 5 cm. vertical laceration, 1 cm. above diaphragm	30 hr.	1. Attempt at repair, catheter in pleural space 2. Open drainage of posterior collection of fluid 3. Jejunostomy	Chronic pleuritis; posterior collection of fluid
	(10) 3.8 cm. linear laceration of left anterolateral wall of lower end of esophagus	12 hr.	Two-layer repair with catheter in pleural space	Foul fluid and cabbage in mediastinum
11. Gardner (1949)[10]	(11) Small hole into the mucosa on the anterior surface	Not stated	Drainage of perforation with Levine tube, via mediastinotomy	Pleural effusion; periesophagitis; esophageal abscess
12, 13. Cliffton (1949)[11]	(12) Longitudinal tear over 4 cm. in length on left posterolateral surface	5½ hr.	Repair of rent with 3 catheters in pleural space	Pleuritis
	(13) No data	No data	Drainage of empyema	No data

One patient reported by Kinsella, Morse, and Hertzog[12] was on the road to recovery when he succumbed to pulmonary embolism on the ninth postoperative day.

due to alcoholism or gluttony; the pain, which is severe and continuous in nature, follows the vomiting rather than precedes it, as in ruptured ulcer and pancreatitis. This lesion should be kept in mind as a postoperative complication in a patient with violent vomiting.[3] Following the acute onset, there is severe prostration with shock and continuous pain, but vomiting usually ceases. The location of the pain, while variable, is usually epigastric initially with radiation to the chest, back, or shoulder, usually on the left side. Thoracic disease may be suggested by the dyspnea, grunting respiration, and cyanosis. Although the initial symptoms may focus attention on the abdomen the abdominal findings do not indicate intra-abdominal disease consistent with the clinical picture. Abdominal tenderness and rigidity may be misleading. However, careful examination of the chest often reveals signs of hydro- or pneumothorax. X-ray examination of the chest is helpful in confirming such observations and in demonstrating air behind the heart. The diagnosis may be proved fluoroscopically by allowing the patient to swallow a small amount of Lipiodol, which leaks into the mediastinum or pleural space. The internist is likely to diagnose these cases as myocardial infarction and the surgeon as perforated peptic ulcer.[2]

In the years 1940 to 1949, when two instances of spontaneous rupture of the esophagus were encountered at the University of Virginia Hospital, 144 cases of perforated peptic ulcer were seen, a ratio of 1 to 72.

Review of the literature has disclosed only thirteen other reports of a successful outcome (see Table I). The ideal treatment of surgical repair without complication following an early diagnosis is exemplified by the case of Olsen and Clagett,[7] in which operation was performed within 3 hours and in which no infection occurred. In other cases the rent was of longer duration and the repair therefore complicated, usually by pleural infection or esophageal fistula. In many of the reported survivals there was either no attempt at primary repair or unsuccessful primary repair with leakage of the esophageal content and resultant empyema or mediastinal abscess.

Although early surgical repair offers the best prognosis, those cases difficult of early diagnosis may be salvaged by appropriate drainage of the pleural space and antibiotic therapy.

SUMMARY

In acute upper abdominal emergencies the possibility of spontaneous rupture of the esophagus should be considered. Evidence of thoracic disease indicates, and Lipiodol swallow establishes, this diagnosis. Early surgical repair is the treatment of choice. A review of cases with successful outcome, together with two new cases, is presented.

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THE INFLUENCE OF CORTISONE ON THE SURVIVAL OF HOMOGRAFTS OF SKIN IN THE RABBIT

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HOMOTRANSPLANTATION of skin appears to be successful only between identical human twins and between litter mates of inbred strains of animals. The failure of skin to persist in the recipient when transplanted from a donor of the same species has not been adequately explained. Experimental studies in rabbits by Medawar[1] and Gibson and Medawar[2] strongly supported the contention that the mechanism by which homografted tissues are destroyed is one of acquired active immunity to the transplanted tissue. On this premise, Medawar has proposed 2 general approaches to the problem: (1) that a donor might be found whose unaltered tissues are compatible with those of the recipient and do not act as an antigen (Longmire and associates[3] found that if compatible skin groups exist in man it is unlikely that they are less than 23 in number) and (2) that the tissues of the graft be altered so that they would no longer be antigenic to the host, or that the immunologic mechanism of the host be altered so that the foreign tissues of the graft would be compatible.

Recent evidence has suggested that compound E, or cortisone, materially alters the immunologic mechanism in animals. It has been demonstrated that lymphoid tissue is concerned with the manufacture and storage of antibodies and that adrenal cortical steroids depress lymphoid tissue and circulating lymphocytes. Harris and Harris[4] noted suppression of the tuberculin reaction in rabbits and guinea pigs treated with cortisone. Germuth and Ottinger[5] found that the Arthus phenomenon was inhibited in rabbits treated with cortisone and that the circulating antibody was suppressed almost 100 per cent. These findings suggested that it would be of interest to study the effect of cortisone in homotransplantation of skin.

METHODS

A mixed stock of 35 pairs of albino rabbits weighing 2 to 3.5 kg. was used. A technique similar to that described by Medawar was followed. Pairs of animals of approximately equal weight that were not litter mates were anesthetized with ether after receiving atropine, $\frac{1}{300}$ gr. intravenously, to prevent accumulation of respiratory secretions. The hair was closely clipped from the left side of the thorax and flank of each animal. The area was prepared with a detergent soap and aqueous Zephiran. With aseptic technique, 6 to 10 pinch grafts of skin were removed from each animal and placed in separate sterile

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Petri dishes containing physiologic saline solution. The grafts were cut approximately equal in size, averaging 1 cm. in diameter. An 8 by 8 cm. square of skin of full thickness was then removed from the same area from which the grafts were taken. The pinch homografts were then placed on the recipient animal in equal numbers and teased into position. A petrolatum gauze dressing covered by sterile gauze was placed over the wound. Plaster of Paris from a 3 inch roll was then applied. The plaster cast was painted with saturated solution of picric acid to prevent the animal from gnawing the dressing. One animal of the pair was given cortisone, 4 mg. per kilogram intramuscularly daily, beginning the day of grafting. The other animal served as the control. The rabbits were anesthetized with ether at intervals of 4 to 6 days, and the grafts were inspected. Grafts were removed for biopsy at appropriate times, and a fresh dressing was applied in the manner described.

RESULTS

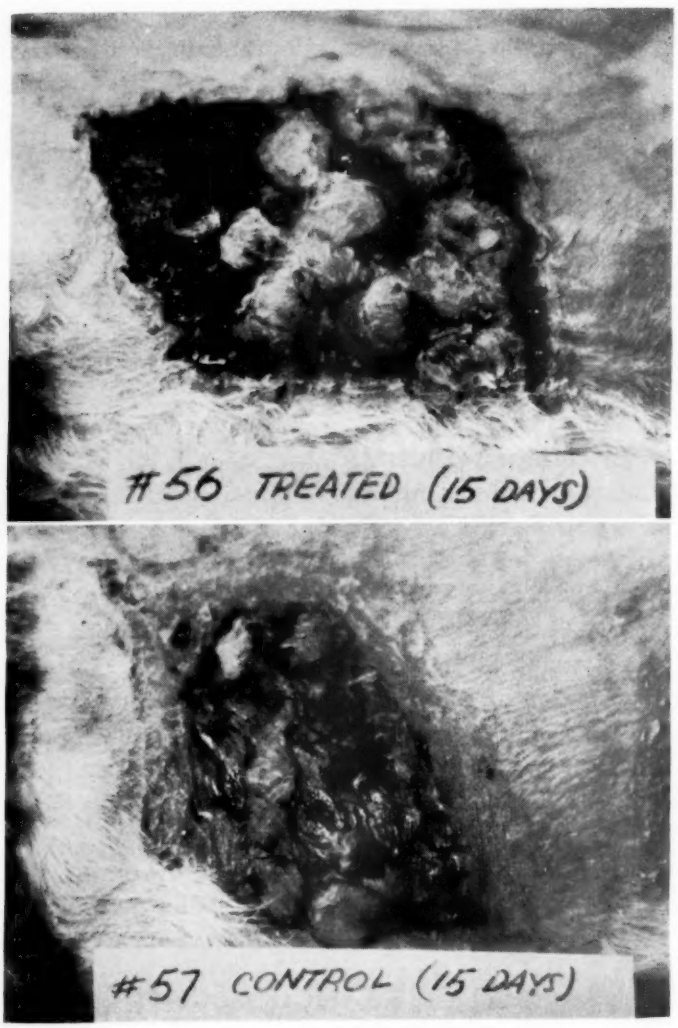
Of the 35 pairs of rabbits, one consecutive series of 6 controls and 6 treated animals succumbed to an epidemic diarrhea within a period of 2 weeks. One pair was lost because of an error in technique when the plaster dressing was improperly applied, and all grafts were lost. Of the remaining 28 pairs, 6 of the cortisone-treated animals died either from diarrhea or pneumonia within 2 weeks, leaving 21 pairs for consideration.

In the 22 untreated control rabbits, all homografts "took" well in the first 4 to 6 days, healing primarily to the graft bed. By the sixth and seventh days, gross evidence of beginning necrosis could be seen. The process of necrosis, as in Medawar's study, appeared complete by the tenth to the twelfth day. Necrotic connective tissue pads often persisted for 3 to 4 weeks before sloughing away or being overgrown by native epithelium and absorbed. Microscopic sections revealed massive invasion of the graft by round cells, a marked proliferation of mesenchymal cells of the host in the region of the graft, eventual obliteration of the vascular channels, and necrosis. These changes were well established by the sixth day and were complete in all instances by the tenth to the fourteenth day.

In 13 of the 22 cortisone-treated animals, the grafts and graft sites presented a marked contrast to the controls in appearance and behavior. Initially all the grafts "took" well, healing primarily to the graft bed, except for an occasional graft accidentally dislodged while the first dressing was being changed. At the initial dressing a considerable difference between treated and control animals was evident in the appearance of the wound. Granulations were very fine and scanty in the treated animals, and the grafts adhered only by a delicate filmy connective tissue. Granulations were more abundant and the grafts more adherent at first on the controls. At subsequent dressings, these differences appeared greater. Granulations remained fine and developed slowly in the cortisone-treated animal. There was little tendency to scarring and contracture, and the decrease in the wound area due to these factors remained at a minimum. In the controls, however, granulations became exuberant, and the wound area decreased greatly in size by contracture of scar tissue.

After 10 to 12 days, when grafts on the controls were completely necrotic, the grafts on the treated animals remained pink and healthy in appearance, and gross evidence of epithelial spread from the margin of the grafts could be seen. After 3 weeks this epithelial spread often extended 0.5 to 1 cm. from the graft margin (see Fig. 1).

A.



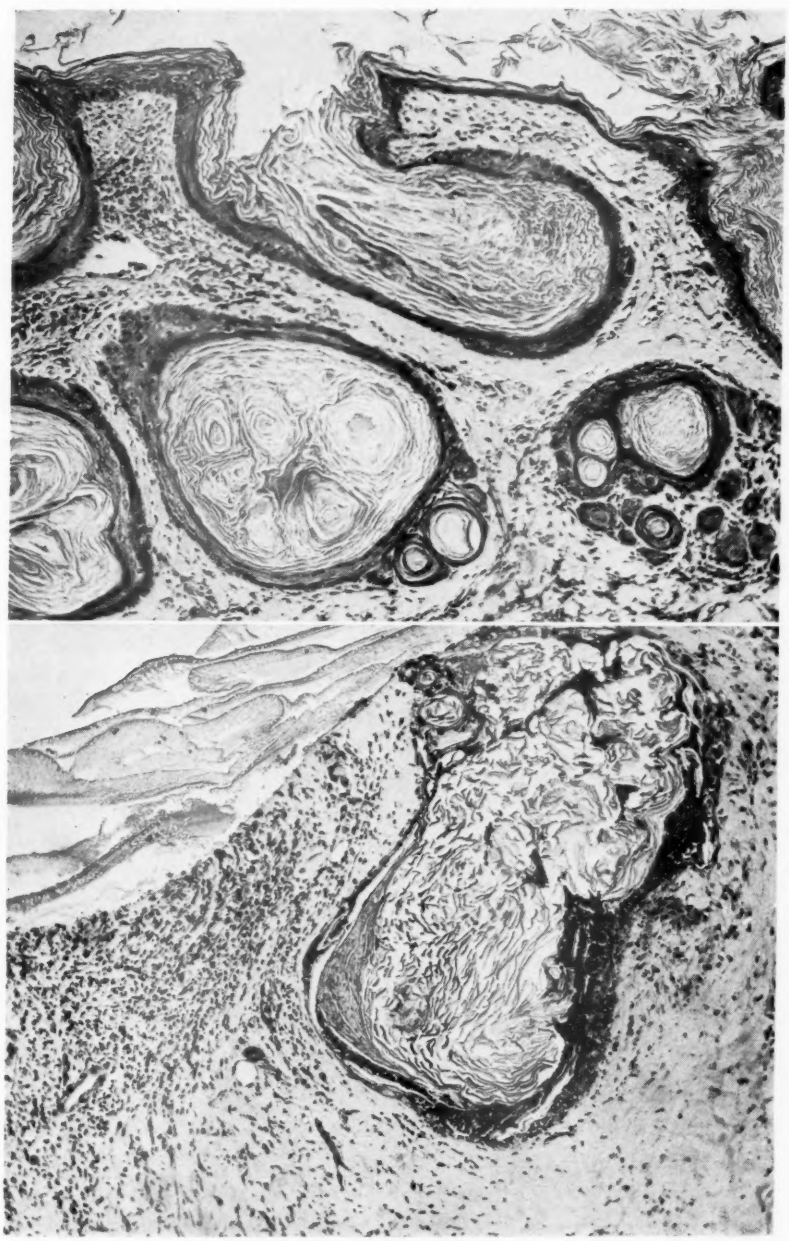
B.

Fig. 1.—Photographs of treated animal and control 15 days following transplantation of skin homografts. Grafts on the control have become hard necrotic crusts. There has been a decided decrease in the wound area by scarring and contracture and by growth of native epithelium from the wound edges. In contrast, grafts on the treated animal are viable and healthy and are surrounded by a halo of epithelium which has spread from the graft margins. In a number of areas they have become confluent. Little or no decrease in the wound area by scar and contracture has occurred.

Microscopic sections of grafts removed from treated animals at 6 to 7 days revealed a rather marked hyaline change and apparent swelling of the connective tissue fibers of the graft. Blood vessels from the host could be

seen invading the graft, but they were not as numerous or as well developed as those in the controls. This was also evident in the amount of bleeding at the time of biopsy. Only a minimal amount of round-cell infiltration was present as a rule. Epithelium was intact over the entire graft in many cases, but

A.



B.

Fig. 2.—Photomicographs of grafts removed for biopsy from a treated animal (*A*) and from a control (*B*) 14 days after transplantation of skin homografts. The Control shows the death of all epithelial elements, only hyaline connective tissue infiltrated with round cells remaining of the graft. Specimen for biopsy from the treated animal shows viable grafts with intact epithelium.

often there was a tendency to loss of epithelium in the graft center, possibly due to lack of nutrition. At the graft margin, however, epithelial elements appeared healthy in all instances and were beginning to spread over adjacent granulations.

Biopsies performed at the end of about 2 weeks showed increased vascularity and persistence of the hyaline change in the connective tissue in the treated animal; the tissue had lost its swollen appearance, however, and appeared more dense. Host connective tissue adjacent to the graft remained loose and fine in character. On the other hand, control grafts, which were now completely necrotic, were avascular and were imbedded in an exceedingly dense connective tissue which the host had formed about them. Again in the treated animals, inflammatory cell infiltration remained at a minimum. Epithelium was intact over the graft in the majority of instances (see Fig. 2), but again loss of epithelium was noted in the center of some of the grafts, particularly the thicker ones. Epithelium at the margins was flourishing, however, and increased spread was evident.

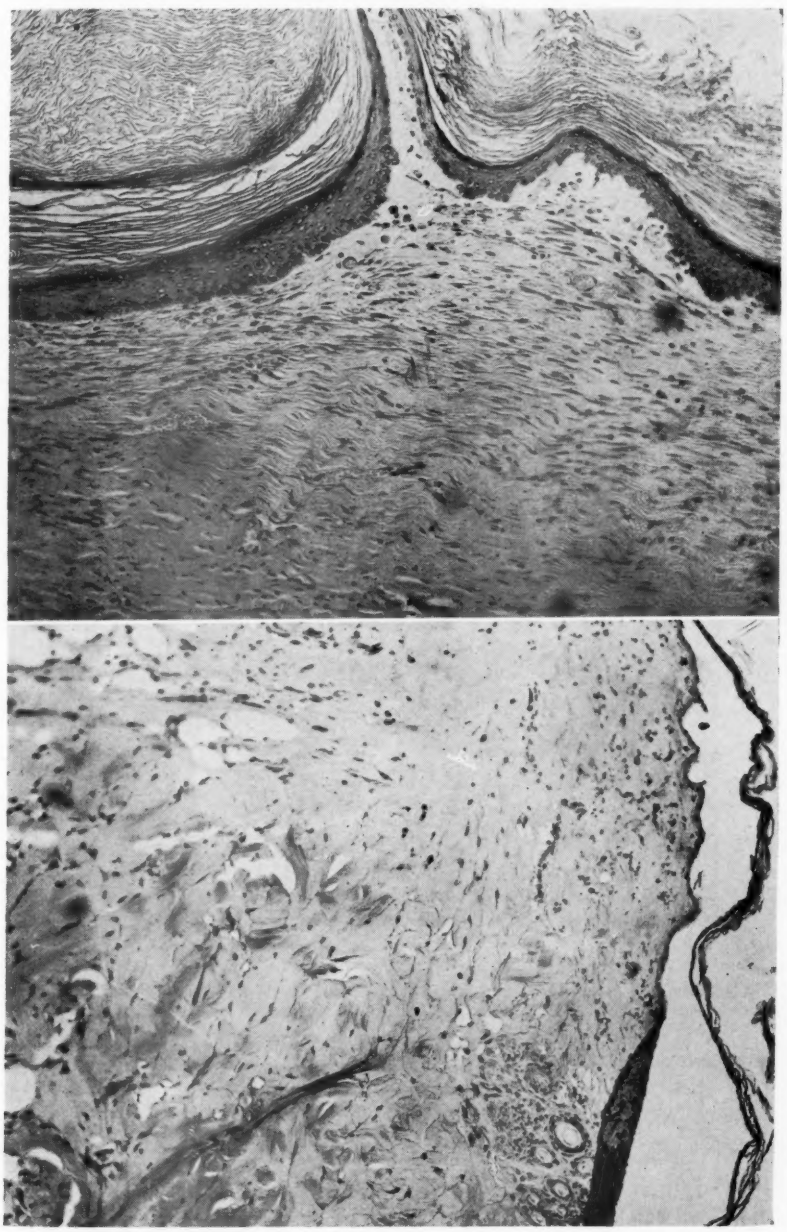
At the end of 3 weeks the epithelial spread was pronounced and was moderately thick near the graft. In thinner grafts, where epithelium persisted at the center, differentiation with formation of apparently new hair shafts was observed. A slight amount of round-cell formation about blood vessels was seen in a number of instances, but otherwise the microscopic appearance of the grafts remained the same (see Fig. 3).

The 13 cortisone-treated animals all lost a considerable amount of weight and apparently became more susceptible to intercurrent infection. Several of the animals died from pneumonia. No more deaths due to pneumonia occurred, however, when penicillin was given routinely at each change of dressing. All other cortisone-treated animals eventually succumbed from a diarrhea. This was presumably of an infectious nature, since it subsequently developed in several of the controls. Stool cultures failed to reveal any bacterial pathogens, and aureomycin brought no improvement in 5 cases in which it was given. Germuth and Ottinger also reported a diarrhea in their cortisone-treated rabbits which they presumed was infectious in origin. They attributed the development of the diarrhea to impairment of the rabbits' natural defenses against disease, having shown that cortisone suppressed antibody formation.

Unfortunately, as a result of the diarrhea, it was impossible to study this group of 13 treated animals beyond 6 weeks. Two were observed 5 to 6 weeks; 5 were observed 4 to 5 weeks. Six died during the third and fourth weeks. Between the beginning of the third week and the end of the fourth, epithelium growing from the graft margins had coalesced. In the fourth and fifth weeks, the wound area was completely epithelized by spread from the grafts and from the wound margins (see Fig. 4). At the end of the fifth week, microscopic sections showed viable grafts much the same in appearance as those seen during the third week, except for the increased thickness and extent of the epithelial spread from the graft border (see Figs. 3 and 5).

The homografts in 2 other cortisone-treated animals behaved much the same in the first 3 week period as those in the group of 13 previously described. All grafts "took" well initially and remained pink and healthy in appearance. A marked spread of epithelium from the graft borders occurred. In the third and fourth weeks the wound area became completely epithelized.

A.



B.

Fig. 3.—Photomicrographs of grafts from treated animal (*A*) and control (*B*) at 24 days and 7 days, respectively. Necrosis of the control graft is well advanced. The spread of epithelium over the host connective tissue from the treated graft is shown in *A*.

However, at this time a pronounced desquamation occurred at the site of each pinch graft over an area approximately the size of the original graft. The desquamating dry scales were undergrown peripherally by epithelium. It appeared that foreign epithelium was gradually being replaced by native epithelium after the wound had been healed as a result of growth from both these sources.

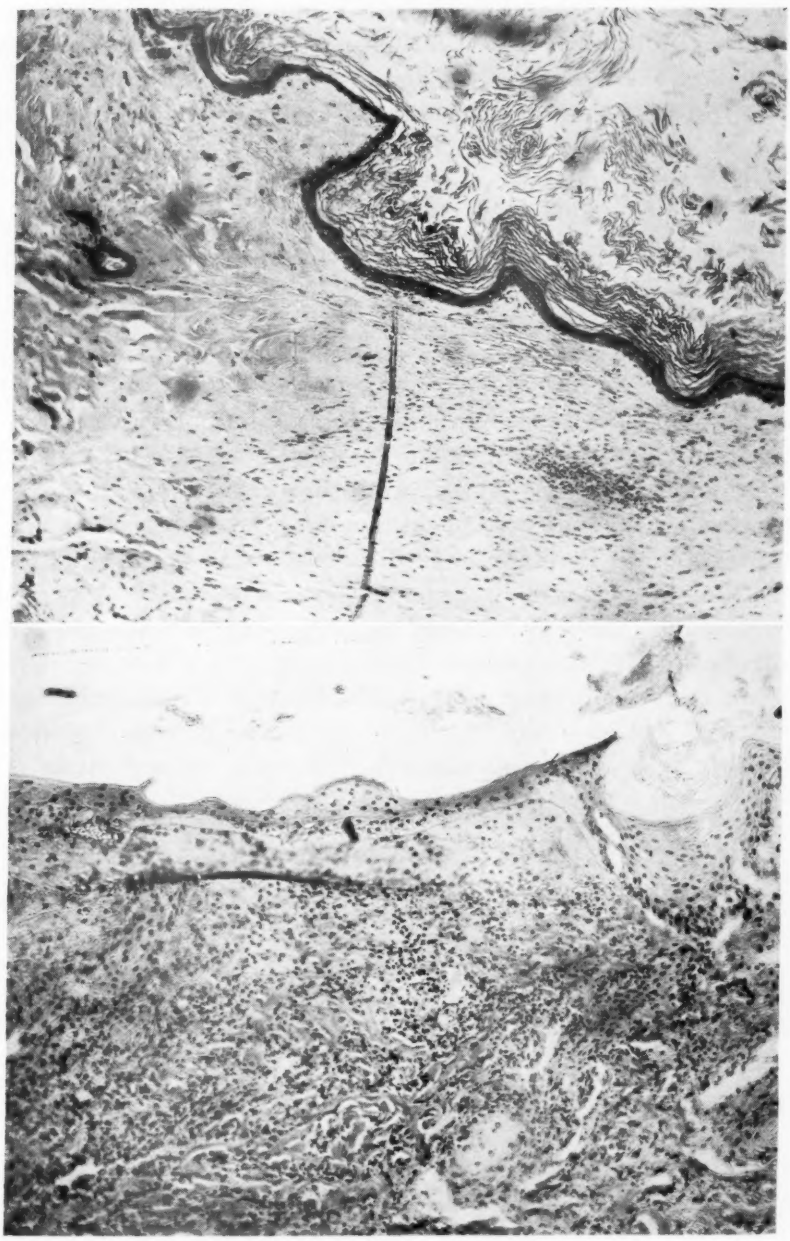
Fig. 4.—Photographs of treated animal (*A*) and control (*B*) 28 days following transplantation of skin homografts. Only dark necrotic pads remain of grafts on the control. Grafts on the treated animal remain viable and have become confluent. Epithelization of the wound, except for the small granulating area, has occurred by spread from the graft margins and by growth of native epithelium from the wound edges.

Cortisone was withheld after the eighteenth day from another treated animal in which the homografts were successful up to that time. The spread of epithelium from the graft border disappeared, and the grafts became necrotic within 6 days.

In the remaining 6 cortisone-treated animals, the homografts did not thrive in the first 3 week period. While destruction of the grafts was noticeably retarded in comparison with that of the controls, necrosis appeared

fairly well advanced at the end of 2 weeks, the grafts appearing dark in color and indurated at this time. Some epithelial spread was noticed grossly in 4 of the animals at an early stage, but this soon disappeared. At the end of

A.



B.

Fig. 5.—Photomicrographs of grafts removed for biopsy from treated animal (*A*) at 28 days and from control (*B*) at 12 days. There is massive invasion of the control graft with round cells. Some epithelial remnants are still recognizable, but appear to be undergoing necrosis. Section in *A* is at the edge of a graft from a treated animal. The graft itself occupies the left one-half of the photograph. Spread of epithelium over the host connective tissue is shown.

the third week, necrosis grossly appeared complete in these animals. Five of this group were old males with very thick skins. In addition, they did not appear to react to the same degree to the cortisone, granulations and contracture of scar tissue in the wound not being delayed to the extent observed in the other treated rabbits.

It is unfortunate that the animals that had apparently been treated successfully could not be observed for a longer period. It is impossible to say at this time whether the destruction of the grafts was merely delayed or whether the grafts would have persisted after the discontinuance of the cortisone. The 2 instances in which desquamation over the graft site occurred during the third or fourth week and the case in which necrosis occurred when cortisone was stopped after the eighteenth day suggest that the life of the homografts is merely prolonged. It is conceivable, however, that if the amount of cortisone were to be gradually diminished after a period of 5 to 6 weeks, an adaptation between the grafts and their new environment might occur.

The suitability of the rabbit as an experimental animal in the problem of skin homografts has been questioned by Rogers[6] because of the existence of the Arthus phenomenon in this animal. It would appear that the breakdown of homografts in the rabbit is exaggerated and accelerated as a result of this phenomenon, a circumstance which would not prevail in other animals or in man. It is possible that the results obtained here with cortisone are due merely to the suppression of this reaction and that they have nothing to do with antibody formation in response to homoantigen. For this reason work is now being conducted on other laboratory animals. In addition, perhaps in more hardy animals the discouragingly high mortality will not be encountered.

The poor results obtained with the thicker grafts and the frequent microscopic finding of loss of epithelium in the graft center in the instances in which marginal epithelium grew well suggest that more success might follow the use of split-thickness grafts.

SUMMARY

1. Homotransplantation of pinch skin grafts in 22 pairs of white albino rabbits was carried out. The technique used was similar to that described by Medawar. One of each pair of rabbits was given cortisone, 4 mg. per kilogram daily, and the other kept as a control.

2. In all 22 control animals, the grafts healed primarily to the graft bed, but necrosis was well under way in many cases by the sixth day and was complete by the tenth to the twelfth day in all cases.

3. In 13 of the treated animals, the destruction of the grafts was definitely delayed at least. The grafts flourished and showed considerable epithelial spread from the graft border.

4. The period of observation of the treated animals was limited, varying from 3 to 6 weeks, because of a susceptibility to a fatal infectious diarrhea.

5. Grossly and microscopically, grafts in the 13 treated animals remained viable, and epithelial spread from the graft continued to occur during the period of observation.

6. In 2 rabbits receiving cortisone, homografts were initially successful. However in the third and fourth weeks, after the denuded area was epithelized, desquamation at the homograft sites occurred, native epithelium apparently replacing that of foreign origin. In another successfully treated animal, the grafts became completely necrotic within 6 days after cortisone was withheld on the eighteenth day.

7. In the other 6 treated animals, although some delay in the destruction of the grafts was evident upon comparison with the controls, necrosis and death of the grafts occurred within 2 to 3 weeks. Five of these failures were in old males with very thick skins.

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CLINICAL AND STATISTICAL STUDIES OF COTTON SUTURE MATERIAL WITH SPECIAL REFERENCE TO COST

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BECAUSE most cotton threads designed specifically for surgical use are relatively expensive, this study was undertaken in order to ascertain whether certain inexpensive cotton threads could be used safely and satisfactorily as suture material.

METHODS

The diameter of various threads studied was determined microscopically with the use of an occular micrometer. Three readings were made on a 1 cm. length of thread and the average was considered as one observation. The average of five such observations on different sections of the thread was taken as the final average diameter.

The breaking points of unknotted five-inch lengths of various threads were determined on spring scales equipped with maximum strain indicators.* A scale with a five-pound capacity was used for the small and medium sizes; a scale with a twenty-pound capacity was used for the large sizes. Dry sand was added at a constant rate until the breaking point was reached. The breaking points of ten consecutive dry, unsterilized five-inch strands were determined for the small and medium sizes (0000, 50, 40, 000, 30, and 00); the breaking points of three strands were determined for the larger sizes. In addition, the breaking points of ten strands of the small sizes (0000 and 50) of cotton threads were determined after autoclaving for thirty minutes at fifteen pounds pressure (tested dry and after soaking in water for a few minutes) and after boiling for ten minutes (tested wet). These threads were wound on rubber tubing for sterilization. Thus a total of forty determinations was made on each of the small size threads.

Average breaking points were calculated and maximum and minimum breaking points noted. From the standpoint of actual usage, the minimum breaking point of a thread is of as much importance as the average breaking point. A thread is actually no stronger than its minimum breaking point and a thread with a low minimum breaking point is difficult to use as a surgical suture.

The dry weight of five yards of most sizes was determined in order to ascertain the actual cotton content of each thread because it was apparent some threads were twisted more tightly than others.

Expensive and inexpensive cotton threads were used in suturing separate halves of the wounds of eighteen patients having first-stage extrapleural thoraco-

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*Manufactured by John Chatillon & Sons, New York, N. Y.

plasties for pulmonary tuberculosis. When the second stage of the thoracoplasty was performed three weeks later, a sample of each thread embedded in muscle was removed. Tissue sections for microscopic examination were made and the reaction of the tissues of the same patient to different cotton sutures was compared.

Inexpensive cotton suture materials were used in a series of seventy-three consecutive patients having 296 thoracoplasty stages.

No attempt was made to investigate differences in the manufacturing processes and the chemical content of the various threads tested as it was considered that such differences were not important if the threads did not produce increased tissue irritation.

The breaking points and diameters of various sizes of one brand of silk suture material were determined for purposes of comparison with cotton.

COTTON SUTURE MATERIAL STUDIED

The cotton suture material studied was as follows:

1. Ethicon surgical cotton sutures manufactured by Johnson & Johnson, New Brunswick, N. J.
2. Seaco surgical cotton sutures manufactured by Seamans and Cobb Company, Hopkinton, Mass.
3. Intrinsic 4 cord left twist, glacé finish cotton thread manufactured for the shoe industry by the American Thread Company, Willimantic, Conn.
4. Geneva 3 cord left twist, glacé finish cotton thread manufactured by the American Thread Company.
5. Regular spool (sewing) cotton manufactured by J. & P. Coates.

COST

The retail prices of the various cotton threads studied as well as packaging details are given in Table I. A tremendous difference in price exists between the inexpensive threads and the one brand of expensive thread listed.

TABLE I. COST OF COTTON THREADS TESTED

THREAD	SIZE	RETAIL PRICE PER 1000 YD.	UNIT SUPPLIED	RETAIL PRICE PER UNIT
Ethicon*	0000	$44.50	100-yard spool	$4.45
	000	44.50	100-yard spool	4.45
	00	56.50	100-yard spool	5.65
	0	68.00	100-yard spool	6.80
	1	68.00	100-yard spool	6.80
	2	79.50	100-yard spool	7.95
Seaco	0000	0.35	6000-yard cone	2.08
	000	0.50	6000-yard cone	3.01
	00	0.70	4800-yard cone	3.33
	0	1.45	1 lb. cone, 2100 yards	3.07
Intrinsic	50	0.31	6000-yard spool	1.83
	40	0.33	6000-yard spool	1.98
Geneva	50	0.35	12 500-yard spools in box	2.10
	40	0.44	12 400-yard spools in box	2.10
J. & P. Coates	50	1.00	50-yard spool	0.05

*Discount of 10 per cent allowed on orders of 12 or more spools which may be assorted as to size.

In 1950, thirty-nine dozen spools of Ethicon cotton costing approximately $2000 were used at Columbia Hospital, a general hospital of 160 beds. The cost of the same quantity of inexpensive cotton thread is about $20.

RESULTS

Results of tests on threads of small and medium size (dry, unsterilized) are given in Table II; results of tests on the large sizes are recorded in Table III. The large sizes are difficult or impossible to break while tying knots during the course of an operation so that the actual breaking points in this group are not important. However, the comparative diameters and textures are of importance in the selection of sutures for the ligation of large blood vessels.

TABLE II. THREADS OF SMALL AND MEDIUM SIZE

	DIAMETER (DRY) (MM.)			BREAKING POINTS (DRY) (POUNDS)					
THREAD	AVERAGE	MAXIMUM	MINIMUM	AVERAGE	MAXIMUM	MINIMUM	DETERMINATIONS	WEIGHT OF 5 YD. (MG.)	MICROSCOPIC APPEARANCE
Ethicon Cotton 0000	0.0514	0.0592	0.0483	2.55	2.75	2.19	10	180	Smooth
Deknatel Silk 0000	0.0520	0.0550	0.0500	2.10	2.12	2.0	3		Slightly rough
J. & P. Coates Cotton 50	0.0560	0.0600	0.0500	2.16	2.37	1.81	10	155	Rough
Intrinsic Cotton 50	0.0565	0.0600	0.0525	2.36	2.56	2.12	10	160	Smooth
Geneva Cotton 50	0.0600	0.0675	0.0550	2.17	2.37	2.06	10	190	Smooth
Intrinsic Cotton 40	0.0625	0.0675	0.0600	2.82	2.94	2.62	10	200	Smooth
Deknatel Silk 000	0.0625	0.0650	0.0600	3.54	3.62	3.5	3		Slightly rough
Geneva Cotton 40	0.0705	0.0775	0.0625	2.98	3.25	2.75	10	230	Smooth
Intrinsic Cotton 30	0.0705	0.0750	0.0625	3.42	3.56	3.19	10	260	Slightly rough
Ethicon Cotton 000	0.0775	0.0858	0.0700	3.85	4.40	3.44	10	250	Smooth
Seaco Cotton 0000	0.0793	0.0891	0.0700	2.39	2.56	2.06	10	150	Rough
Ethicon Cotton 00	0.0827	0.0833	0.0808	4.76	5.25	4.19	10	400	Smooth
Deknatel Silk 00	0.0925	0.1000	0.0850	5.0	5.0	5.0	3		Rough
Seaco Cotton 000	0.1065	0.1200	0.0925	4.33	5.25	4.00	10	310	Very rough

The breaking points of the small sizes of threads after autoclaving (tested dry and wet) and after boiling (tested wet) are given in Table IV.

In the group of small sutures (0000 and 50) most commonly used for ligating small vessels, Ethicon 0000 was found to be the strongest and finest. However, Intrinsic 50 is 95 per cent as strong, has a diameter only 8 per cent larger, and contains slightly less cotton by weight (dry, unsterilized). There was little difference between the minimum breaking points of these two threads

TABLE III. THREADS OF LARGE SIZE

THREAD	DIAMETER (DRY) (MM.) AVERAGE	MAXIMUM	MINIMUM	BREAKING POINTS (DRY) (POUNDS) AVERAGE OF 3 DETERMINATIONS	WEIGHT OF 5 YARDS (MG.)	MICROSCOPIC APPEARANCE
Ethicon Cotton 0	0.1200	0.1300	0.1125	7.37	650	Smooth
Ethicon Cotton 1	0.1270	0.1275	0.1250	8.85	760	Slightly rough
Seaco Cotton 00	0.1315	0.1433	0.1242	5.96	470	Very rough
Deknatel Silk 0	0.1375	0.2125	0.1125	7.00		Slightly rough
Deknatel Silk 1	0.1455	0.1525	0.1375	10.33		Smooth
Deknatel Silk 2	0.1480	0.1500	0.1450	10.25		Smooth
Ethicon Cotton 2	0.1500	0.1650	0.1325	9.25	970	Slightly rough
Deknatel Silk 3	0.1635	0.1750	0.1550	14.17		Smooth
Seaco Cotton 0	0.2125	0.2342	0.1917	12.41	1050	Very rough

when dry (unsterilized and after autoclaving) and when wet (after autoclaving and after boiling).

Although the dry diameter of Seaco 0000 is quite large, it is loosely twisted and actually contains less cotton than any of the other threads tested. The diameter of this thread when wet compares favorably with those of the other threads of small size. The average and minimum breaking points of Seaco 0000 are fairly satisfactory but it is a rough thread (Fig. 1). When knots are tied in rough threads of small size, these threads have a tendency to fray and break easily as a result of this fraying. This tendency to fray is somewhat less noticeable when the thread is wet.

Ordinary 50 spool (sewing) had the lowest average and minimum breaking points of any of the cotton threads tested. It is also a rough thread.

It will be seen in Table II that Intrinsic 40 and Geneva 40 are actually intermediate sizes between Ethicon 0000 and Ethicon 000. The average and minimum breaking points of Intrinsic 40 and Geneva 40 are satisfactory considering their diameters and cotton content. These latter two threads can be used for suturing muscle and fascia. Ethicon 000 should be compared with Intrinsic 30 rather than with Intrinsic 40 and Geneva 40.

It will be noted that the dry diameter of Seaco 000 is greater than that of Ethicon 00 although its weight is less. Seaco 000 is almost as strong as Ethicon 00 and should be compared with this suture rather than with Ethicon 000.

As noted before, the large sutures listed in Table III are used principally for the ligation of large vessels. We have found that there is an advantage in using a suture with a rough texture for the ligation of vessels in a deep wound

TABLE IV. STERILIZATION OF THREADS OF SMALL SIZE

THREAD	BREAKING POINTS IN POUNDS (10 DETERMINATIONS)										DIAMETER (MM.)		MICROSCOPIC APPEARANCE
	DRY UNSTERILIZED (AVERAGE)	AUTOCLAVED DRY			AUTOCLAVED, THEN WET			BOILED, KEPT WET			DRY UNSTERILIZED (AVERAGE)	BOILED, KEPT WET (AVERAGE)	
		AVERAGE	MAXIMUM	MINIMUM	AVERAGE	MAXIMUM	MINIMUM	AVERAGE	MAXIMUM	MINIMUM			
Ethicon 0000	2.55	2.50	2.81	2.00	2.88	3.12	2.50	2.72	3.12	2.50	0.0514	0.0735	Smooth
Intrinsic 50	2.36	2.33	2.56	2.12	2.62	2.87	2.37	2.67	2.81	2.44	0.0565	0.0675	Slightly rough
Geneva 50	2.17	2.07	2.37	1.87	2.30	2.37	2.19	2.34	2.87	2.12	0.0600	0.0757	Slightly rough
Seaco 0000	2.39	2.38	2.50	2.19	2.62	2.94	2.25	2.44	2.81	2.25	0.0793	0.0703	Slightly rough

(such as in a pulmonary resection) because the first knot will not tend to slip as it sometimes does when a smooth suture is used. We have found Seaco 00 and 0 satisfactory ligatures for large vessels.

It will be seen in Table IV that the autoclaving weakens threads slightly when tested dry. Wet threads are definitely stronger than dry threads even after sterilization. When a thread is tested wet, it has the same tensile strength whether autoclaved or boiled.

The results of microscopic examinations of muscle tissues containing expensive and inexpensive cotton suture material from the same patient follow. Comparison of Ethicon 000 with Seaco 000 in seven patients consistently showed slightly more tissue reaction around the Seaco sutures. However, since this increase was slight, it is possibly not significant. Comparison of Ethicon 000 with Intrinsic 40 in six patients and Geneva 40 in 5 patients failed to show any appreciable difference in tissue reaction around these sutures.

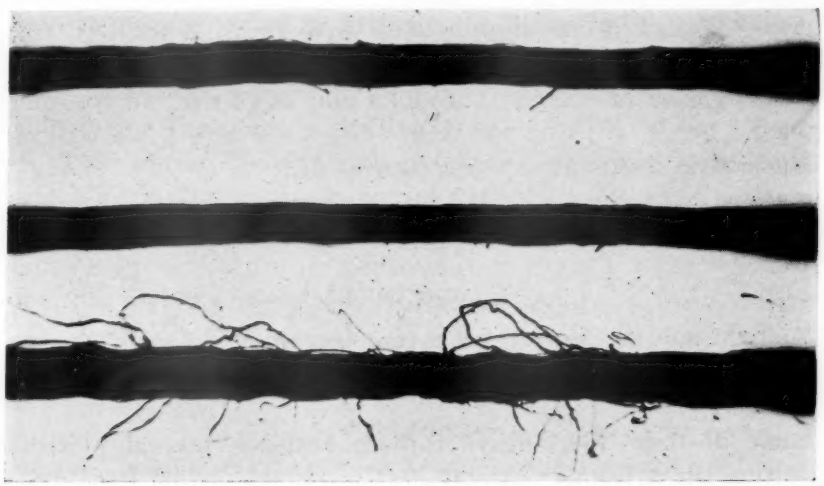

Fig. 1.—Microscopic appearance (×110) of 3 cotton threads tested. Top, Ethicon 0000; middle, Intrinsic 50; bottom, Seaco 0000.

In the seventy-three patients having 296 thoracoplasty stages during which various cotton sutures were used, there were no significant wound infections. The incision of one patient opened in several places four weeks following his last stage operation and cotton (Seaco) suture material was extruded. In these 296 thoracoplasty stages the type of suture materials used was as follows: Seaco only, 233 stages; Geneva only, 18 stages; Intrinsic only, 14 stages; Ethicon only, 4 stages; Seaco and Ethicon, 10 stages; Geneva and Ethicon, 7 stages; Intrinsic and Seaco, 5 stages; Intrinsic and Ethicon, 4 stages; Geneva and Seaco, 1 stage. The same suture materials were not always used at all stages on the same patient.

DISCUSSION AND RECOMMENDATIONS

Although Ethicon cotton was found to be slightly stronger in relation to its diameter than the inexpensive threads tested, this advantage is so small that it is insignificant from a practical standpoint. Furthermore, in relation to

cotton content rather than to diameter, some of the inexpensive threads of small size are as strong as Ethicon.

There was no more tissue reaction to Intrinsic 40 and Geneva 40 than to Ethicon 000. Although there was slightly more tissue reaction to Seaco 000, this was so slight that it is possibly not significant. Furthermore, Seaco was used as the only suture material in 233 thoracoplasty stages and was extruded later in only one patient.

Since the tensile strength of cotton thread is definitely greater when wet, it is recommended that cotton sutures always be used wet in order to take advantage of this added strength. Cotton sutures may be sterilized by either autoclaving or boiling if used wet.

For the ligation of small vessels, we recommend the use of Intrinsic 50 cotton thread. For suturing muscle and fascia, we recommend either Intrinsic 40 or Geneva 40. If stronger sutures are desired for fascia, Intrinsic 30 or Seaco 000 may be used.

Intrinsic 40 and 50 are slightly stiff when dry. When wet, this stiffness disappears.

For the ligation of vessels of medium and large size, we recommend Seaco 000, 00, or 0.

Intrinsic 30 is a satisfactory skin suture.

Our actual experience with the use of these inexpensive cotton suture materials supports the conclusions drawn from our statistical studies.

CONCLUSIONS

1. Some brands of inexpensive cotton thread are as satisfactory for use as surgical suture material as expensive thread manufactured specifically for surgical use.

2. Some of these inexpensive threads compare favorably with the more expensive threads as far as diameter, cotton content, breaking points, and tissue reaction are concerned.

3. There is a tremendous difference in price between the expensive and inexpensive threads tested.

Case Reports

HEMANGIOPERICYTOMA OF THE SIGMOID COLON

Case Report

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In 1942 Murray and Stout[1, 2] introduced the term hemangiopericytoma to describe a tumor arising from the cells of the outer walls of capillaries. These cells had been named pericytes in 1923 by the Swiss histologist, Zimmerman.[3] There have been other reports of this tumor.[4-9]

This report will be largely concerned with the clinical course of a patient whose sigmoid tumor (hemangiopericytoma) was resected by one of us (G. W. A.) in 1946. This will be done in some detail for there have been no previous reports on the bizarre clinical development of this lesion in the colon.

We do not feel qualified to discuss the histology of these tumors but take this occasion to suggest that future studies follow Stout's[7] suggestions. He has recommended that sections be stained with hematoxylin and eosin and also with Laidlaw's silver reticulin stain, and that the magnification for photomicrographic reproduction be 515 diameters.

HEMANGIOPERICYTOMA

There is not sufficient recorded evidence to indicate that hemangiopericytomas may be recognized by their gross appearance. To date, the final diagnosis has been made on the histologic appearance of the lesion. Our experience with the gross characteristics of this lesion is confined to this one case. However, we would like to confirm the opinions expressed elsewhere that the tumor is nearly always a hard, irregular, nodular, fibrosclerotic lesion rarely confined by a definite capsule. It is slowly progressive and its invasion of contiguous tissues and structures is noteworthy. Extreme vascularity, such as encountered in the angiomas and nevi, is unreported. Our experience at the second operation of Oct. 9, 1948, indicates that the vascularity of these lesions may be dependent upon their proximity to large vessels and not upon the inherent structure of the tumor. The rate of growth of the initial lesion and the metastasis has been most variable and difficult to evaluate. The majority of cases have been located in the superficial soft tissues. Others have been located within the thorax, and abdominal and pelvic cavities. Involvement of the jejunal mesentery,[7] ileum,[7] and omentum[6] have been reported. There have been no previous reports on the clinical development of this lesion in the colon.

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CASE REPORT

A 34-year-old white nulliparous woman weighing 134 pounds was seen on Jan. 9, 1946, complaining of intermittent periods of colicky abdominal cramping associated with the frequent discharge of mucus and dark and fresh blood by anus during the pervious two months. Barium enema and gastrointestinal series on Jan. 3, 1946, showed a defect in the upper sigmoid. The past history was irrelevant except for a supracervical hysterectomy, bilateral salpingo-oophorectomy, and appendectomy that had been done elsewhere in 1943. This tissue was re-examined at our request and microscopy revealed fibroid, hemorrhagic corpus luteum cyst and chronic appendicitis. There was no evidence of endometriosis. Progynon B had been taken fairly regularly since 1943 to relieve menopausal symptoms. At examination the patient was found to be well nourished and not obstructed. The positive findings were limited to visualization of fresh and dark blood and mucus during proctoscopy to 12 inches. Abdominal and pelvic examination was unsatisfactory. The diagnosis was carcinoma or polyp of the upper sigmoid colon.

Jan. 17, 1946, a resection of 6 inches of the upper sigmoid colon with end-to-end anastomosis was done. Operative notes were abstracted as follows: The abdominal viscera and liver were normal. There were no demonstrable glands along the aorta or in the root of the mesentery at or near the inferior mesenteric vessels. There was a hard, fibrosclerotic, irregular, nodular, intramural, polypoid-like tumor approximately 1½ by 1 inch in size involving the sigmoid colon. About one-half of it extended into the mesocolon and the other one-half partially occluded the sigmoid lumen by invasion and obturation. It had the gross appearance of a sarcoma. It was grayish-white and was mottled or trabeculated with brown splotches and filaments which appeared like pigment. Two discrete glands in the adjacent mesentery appeared the same. It was opened (after operation) and the overlying mucosa was eroded and bleeding. No fungating ulcerative lesion was seen. It was sent to the laboratory with a diagnosis of sarcoma. The postoperative course was uneventful.

From February, 1946, to July, 1947, there were intermittent periods of dull right lower quadrant aching and discomfort which were referred to the right hip, gluteus, and renal area. These symptoms were thought to be due to endometriosis. They were partially relieved by Progynon B given elsewhere. There were no further bowel symptoms.

On Aug. 22, 1947, a right nephrectomy for nonspecific unilateral pyelonephritis was performed elsewhere. Microscopic examination showed extreme hydronephrosis with slight dilatation of ureter.

Between Nov. 17, 1947, and Oct. 9, 1948, abdominopelvic and proctoscopic examinations on three occasions revealed a hard, irregular, fixed, flat mass approximately 4 by 6 inches in circumference occupying the right pelvis. This growth progressed slowly and was considered to be a retroperitoneal endometriosis influenced by Progynon B.

On Oct. 9, 1948, exploration of the abdominopelvic cavity revealed a normal liver and no abnormal glands along the aorta. Operative notes were abstracted as follows: An irregular, hard, grayish-white mass 1½ inches in size was present in the mesosigmoid near the site of the previous resection. Recurrence of the hemangiopericytoma was diagnosed and the mass was sent to the laboratory. Pelvic exploration did not reveal any evidence of endometriosis. A hard, nodular, irregular, retroperitoneal mass was infiltrating the right lesser pelvis. It involved the internal iliacs, right ureterovesical area, right broad ligament, and adjacent cervix. It was a broad, flat, irregular, stony hard, immobile lesion 2 to 3 inches in diameter. The rectum was displaced slightly to the left. Resection was attempted following ligation and dissection of the internal iliacs. Right hemicystectomy, with the plane of dissection to include the upper third of the vagina, cervix, right broad ligament, and ureter, was done. The slow tedious dissection was abandoned at the end of five hours due to the inability to elevate and ligate the large gluteal vessels with collaterals. These were visualized, but the mass could not be mobilized and exsanguinating hemorrhage was anticipated if ligation was not done before further dissection. The cervix, upper one-third of the vagina,

and the broad ligament were removed with part of the tumor mass. The bladder was reconstructed. The lesion was considered unresectable.

This patient was seen intermittently by us until July, 1949. She resumed her occupation and her family physician stated in January, 1951, that she remains in fair health, without too great a disability. Total time of observation and care was 5 years (January, 1946, to January, 1951).

Microscopic Pathology.—The microscopic report* (No. 16492) on the sigmoid colon tumor resected Jan. 17, 1946, was as follows: Sections from the tumor showed a very richly cellular stroma in which there was no particular definite pattern. The cells were round for the most part, but in many instances assumed a bizarre fusiform shape. The nuclei were hyperchromatic, and many mitotic figures were noted. The stroma was highly vascular and many new-formed blood vessels were seen, some of which showed compression and distortion.

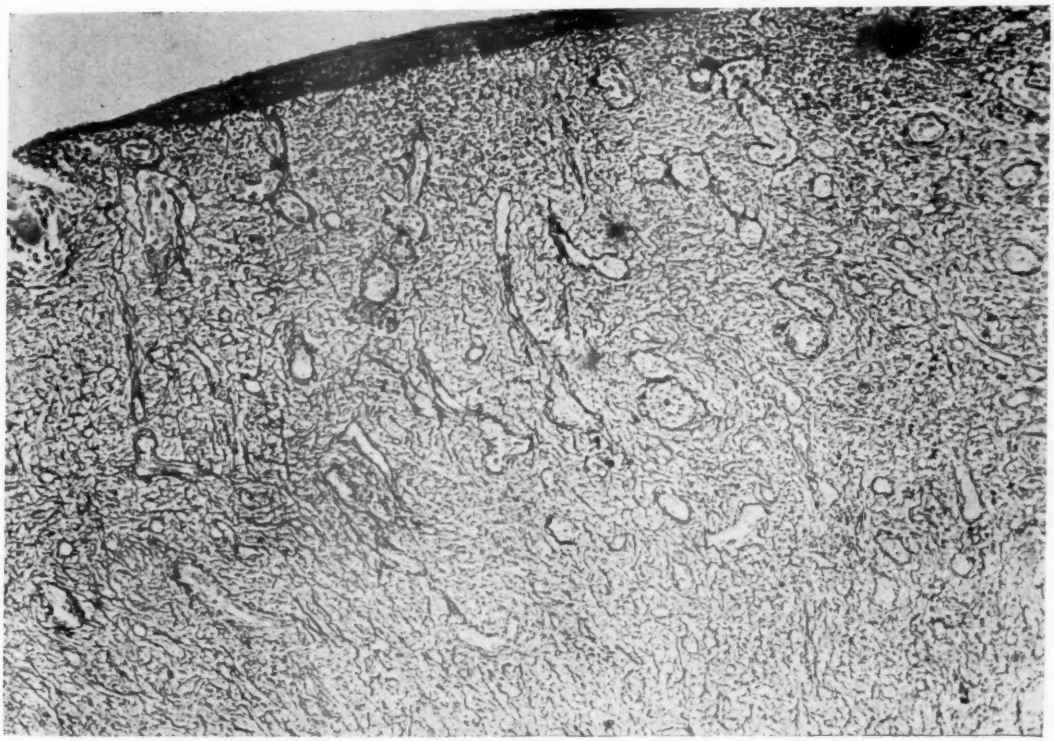

Fig. 1.—Tumor of sigmoid colon (hematoxylin and eosin stain, ×125). (United States Army Medical Museum No. 93593.)

The growth had extended through the wall of the bowel involving all coats, and in one portion involved the mesocolon. Sections from the small gland submitted showed no evidence of malignant involvement. The picture was that of a hemangiopericytoma arising from the pericytes of the capillaries and being of a relatively low grade malignancy. Consultation on this tissue at the United States Army Institute of Pathology confirmed the diagnosis.

The microscopic report† (No. 48s3844) on the pelvic tumor partially removed Oct. 9, 1948, was as follows: Sections taken from the pelvic tumor showed angioblastic tissue embedded in a dense fibrous stroma. Numerous capillaries with definite endothelial walls and numerous capillary-like spaces were surrounded by a proliferation of endothelial-like cells. The individual tumor cells had dense, oval- or spindle-shaped nuclei and had a scant amount of cytoplasm occasionally associated with collagenous-like processes. There was an occasional binucleated form which stained deeply with hematoxylin. The proliferation of

*By Dr. O. B. Hunter, Director of Pathology, Doctors Hospital.

†By Dr. Charles F. Geschickter, Professor of Pathology, Georgetown University School of Medicine.

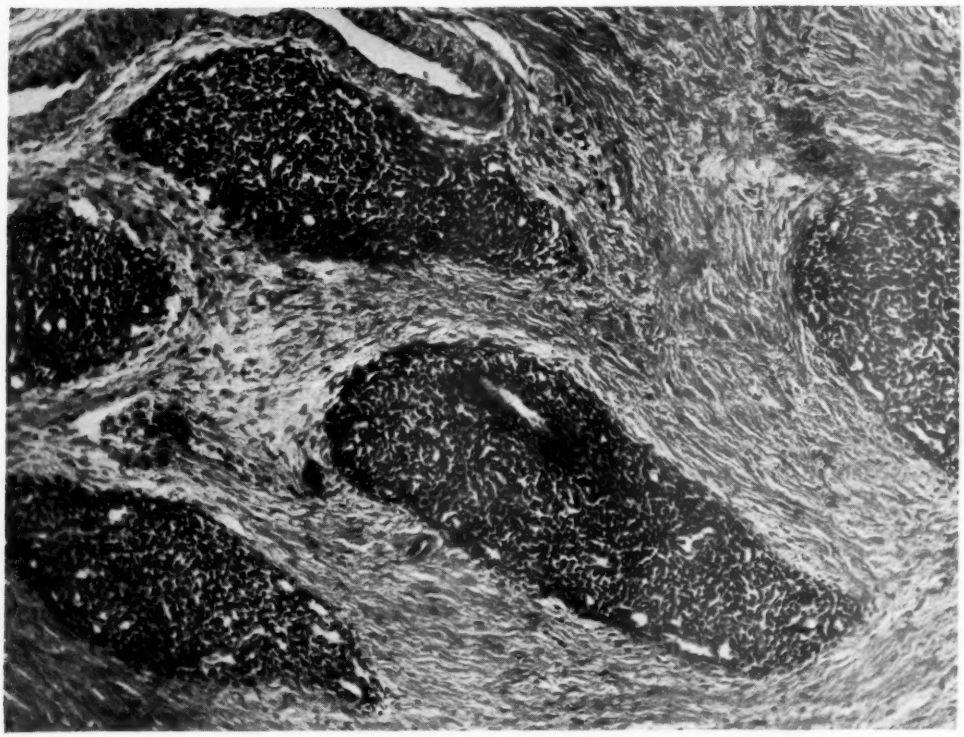

Fig. 2.—Dense fibrous stroma showing islands of neoplastic cells, one of which is adjacent to an arteriole. The cells present no particular pattern but are arranged in some areas concentrically to blood spaces. The cells are round or ovoid in shape and there is a rather well-defined uniformity in size. The nuclei are moderately hyperchromatic and an occasional mitotic figure can be seen (hematoxylin and eosin stain, ×145).

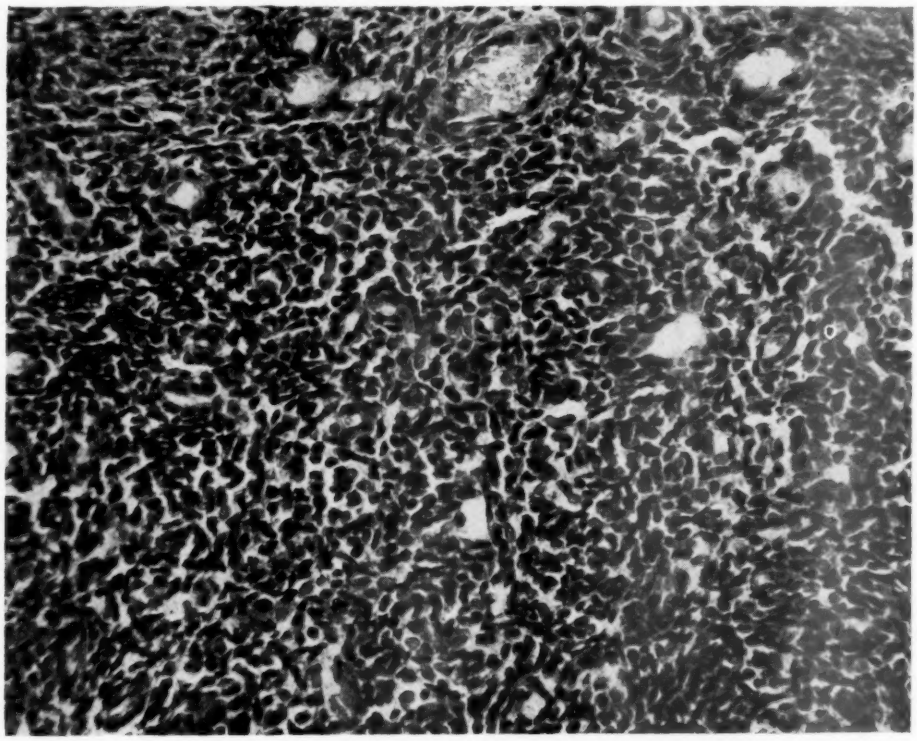

Fig. 3.—Same as Fig. 2, but higher magnification (hematoxylin and eosin stain, ×315).

capillaries and endothelial-like cells was characteristic of the so-called benign angioblastomas found in the skin of infants. The presence of such tissue in an adult and invading fibrous tissue is indicative of malignancy and characteristic of the hemangiopericytoma described by Stout. The endothelial-like cells proliferating about the capillaries and capillary spaces were primitive mesenchyme or pericytes capable of forming both fibrous tissue and smooth muscle. The solid tumor from the sigmoid mesentery was beneath the peritoneal surface and showed the same histology as the pelvic tumor. The diagnosis was hemangiopericytoma.

DISCUSSION

This case is reported as hemangiopericytoma of the sigmoid colon, for it was in this location that the first tumor mass was discovered. The gross appearance is given in detail for there is scant information available in this respect. It is interesting to note that the intestinal symptoms and findings in January, 1946, obscured the symptoms starting in February, 1946. These latter were intermittent periods of dull right lower quadrant aching and discomfort referred to the right hip, gluteus and renal areas. They have persisted until she was last seen in January, 1951. These symptoms were responsible for the urologic studies revealing a low enlarged right kidney which was removed in August, 1947, with only partial relief of the symptoms. The dull, intermittent aching and discomfort referred to the right hip, gluteus and right lower quadrant were thought to be of endometrial origin. This feeling was based on the chronicity, physical character of the palpable mass, and the variable response to Progynon B. The true nature of the deep pelvic tumor located flat against the right pelvis was not revealed until the second operation of October, 1948.

This patient's five-year bizarre clinical course is indicative of the difficulties one may encounter with any rare slow-growing tumor. The pelvic tumor mass was difficult to palpate by rectum and relatively inaccessible upon vaginal examination despite frequent examinations and consultations.

SUMMARY

1. A case of hemangiopericytoma of the sigmoid colon is presented in clinical detail.

2. Some of the features of this rare tumor are discussed.

3. We wish to recommend that further clinical studies of this type of lesion be sought out and reported as they become recognized.

We are indebted to Dr. Milton Flocks for permission to publish this case.

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GASTROILEOSTOMY AND GASTROCOLOSTOMY

Report of Three Cases

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ALTHOUGH gastroileostomy and gastrocolostomy are rare surgical errors which result from improper identification of the intestine in performing gastroenteric anastomosis, two such cases have been observed during the past three years, and a third was discovered in a review of hospital records. These cases are presented in order to call attention again to this unfortunate condition.

REPORT OF CASES

Case 1.—A. A. C., a 25-year-old man, was admitted to Touro Infirmary on June 8, 1936. Three years before he had been operated upon elsewhere for a bleeding peptic ulcer, and the food passage had been changed. A few days postoperatively, after administration of a soapsuds enema, the patient vomited and complained of tasting the soapsuds. Shortly thereafter, he began to have lienteric stools approximately one-half hour after meals. Fifteeen to twenty bowel movements occurred daily until two weeks before admission, when he experienced inability to defecate, became nauseated, and vomited. Formed stools had been noted in the vomitus. He had lost 30 pounds during the three years before admission.

Except for evidences of malnutrition, no significant physical abnormalities were noted. Roentgenography following a barium meal revealed a gastrocolic fistula between the greater curvature of the stomach and the mid-transverse colon (Fig. 1, *A*).

At operation on June 15, 1936, the transverse colon was found to be anastomosed to the posterior wall of the stomach, and an old healed duodenal ulcer was present. After the omentum had been separated, the gastric portion of the anastomosis was divided and the stomach closed in three layers. The transverse mesocolon contained such large, hard nodes that carcinoma was suspected. Therefore, this portion was resected and end-to-end anastomosis performed. Examination of the specimen revealed an enormous ulcer in the colon opposite the anastomosis (Fig. 1, *B*). The postoperative course was uneventful, and the patient was discharged July 2, 1936. There has been no further contact with the patient.

Case 2.—J. W., a 34-year-old physician, was admitted to Foundation Hospital, Jan. 22, 1947. He had had recurrent exacerbations of a duodenal ulcer for many years, and a crater had been demonstrated roentgenographically. Seven months previously, gastric resection had been performed elsewhere. Since then, the patient had had four or five stools daily. Two episodes of more severe diarrhea, occasionally lienteric, had occurred. He had lost 50 pounds. Roentgenograms made elsewhere established the diagnosis of gastroileostomy. The surgeon who performed the original operation recalled that the proximal jejunum had been identified but was replaced momentarily. Subsequently, delivery and anastomosis of what was thought to be the same loop proved to be a loop of the terminal ileum.

Physical examination revealed only two small superficial draining sinuses in the upper midline scar. Values of formed elements of the blood, chloride, carbon dioxide combining power, and proteins were not significantly altered.

On Jan. 25, 1947, exploration through a left paramedian incision revealed that about one-third of the stomach had been resected and the end of the stomach had been anastomosed to the side of the terminal ileum about twelve inches above the ileocecal valve. This anastomo-

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sis was separated, and after transverse closure of the ileum, about 90 per cent of the stomach was resected according to the Hofmeister-Pólya technique. A double jejunostomy was performed just distal to the stoma; one tube was inserted into the stomach and the other directed into the distal jejunum to be used for feeding purposes.

Fig. 1,*A* (Case 1).—Roentgenogram after barium meal. Note the passage of almost all of the barium directly into the transverse colon.

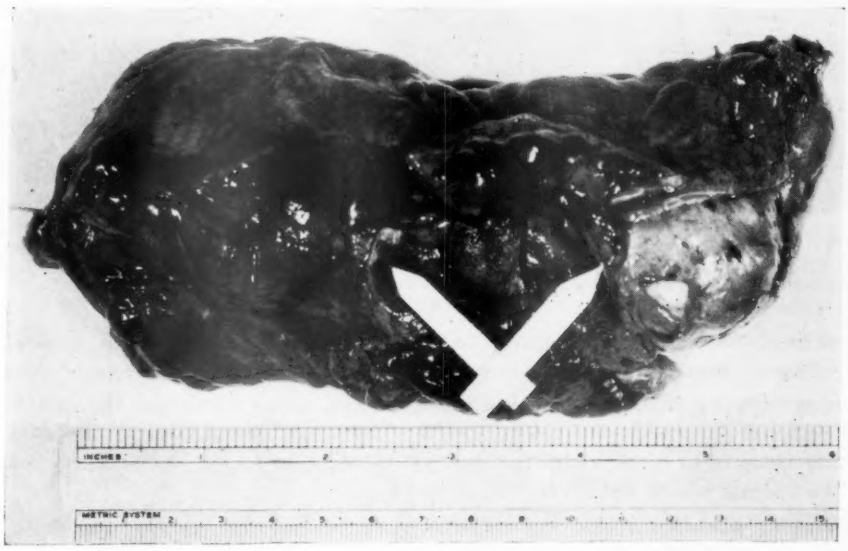

Fig. 1,*B* (Case 1).—Resected segment of transverse colon. The arrows point to the proximal and distal openings through the stoma. An ulcer is opposite, and the mesocolon is greatly indurated.

Jejunal feedings were started on the second postoperative day, and the patient began to take a soft diet on the seventh day. However, on the tenth day he began to have five to six stools daily and postprandial cramps. Since then he has been incapacitated to a varying degree by a dumping syndrome and inability to gain weight.

CASE 3.—Mrs. F. L. C., a 43-year-old woman, was admitted to Foundation Hospital on May 21, 1950. She had had several laparotomies for removal of the pelvic organs and subsequent lysis of adhesions. Ten months before admission, partial gastric resection had been performed elsewhere for abdominal pain, nausea, and vomiting. Persistence of symptoms one month later led to another laparotomy for lysis of adhesions. Since then, she had had recurrent episodes of nausea, vomiting, diarrhea, and pain in the epigastrium and right lower abdominal quadrant. Diarrhea with dark green to black stools had been persistent, and the patient lost 60 pounds in weight during the eight months prior to admission.

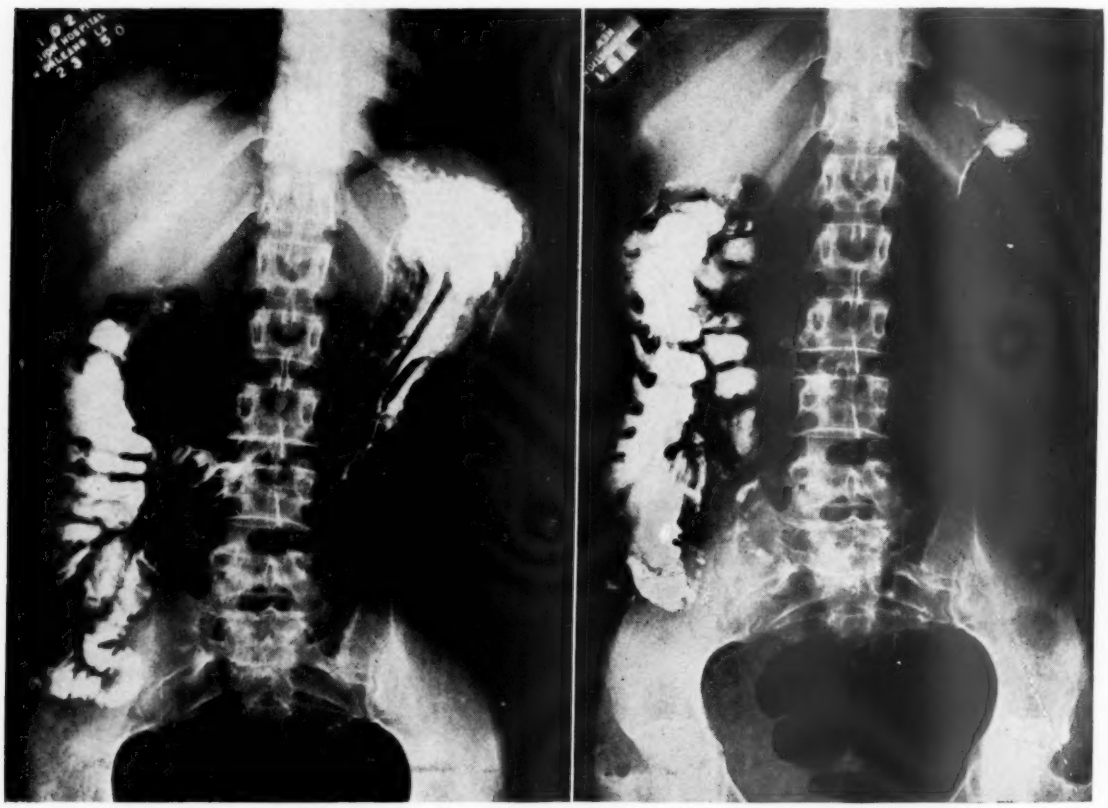

Fig. 2 (Case 3)—*A*, Roentgenogram after barium swallow. Anastomosis demonstrated in terminal ileum and large amount of barium has already entered the ascending colon. *B*, Film thirty minutes after barium swallow showing almost all of the barium in the colon.

Examination revealed a dehydrated, extremely emaciated patient with cheilosis and a red, beefy tongue. Some tenderness in the upper abdominal region was present.

Roentgenography following a barium meal revealed that a portion of the gastric antrum had been resected and end-to-end anastomosis had been established between the stomach and the terminal ileum, 6 to 8 cm. above the ileocecal valve (Fig. 2, *A*). After thirty minutes almost all the barium was in the ascending colon (Fig. 2, *B*).

Laboratory studies revealed the hemoglobin to be 14.2 Gm. or 91.5 per cent, and the hematocrit was 49 per cent. Chlorides were 92 meq. per liter and total proteins 4.9 Gm. The electrocardiogram showed changes indicative of hypokalemia.

Preoperative preparation consisted of hydration and restoration of electrolyte balance, correction of the vitamin deficiency, and restoration of a depleted blood volume and proteins.

At operation on May 29, 1950, multiple adhesions were divided and the gastroileostomy separated. The ileum was closed transversely and four-fifths of the remaining stomach resected according to the Hofmeister-Pólya technique. A Witzel-type jejunostomy was made 10 cm. distal to the stoma for feeding purposes.

Tap water feedings through the jejunostomy tube were begun on the first postoperative day, followed the next day by a dilute jejunal formula. The amount and concentration of this were increased rapidly until the patient was tolerating an almost continuous drip of full-strength formula on the fifth postoperative day. Oral feedings were started on the fifth day and gradually increased until an adequate diet was tolerated by the twelfth postoperative day. The jejunal tube was removed three days later with prompt healing of the wound. The patient was dismissed on the twentieth postoperative day with instructions to take an antispasmodic for mild abdominal pain.

Since dismissal, the patient has gained about 40 pounds in weight and, except for persistent headaches and mild intolerance to fatty foods, she has been well.

DISCUSSION

Of course, gastroileostomy should never occur, but even experienced surgeons have been known to commit or narrowly avert this error. It is not difficult for a loop of ileum to displace the previously identified first loop of jejunum if it is momentarily replaced. This possibility is usually not considered at the time, and the differentiating features between jejunum and ileum are not observed. However, if a clamp or suture is placed on the jejunum after identification of the ligament of Treitz, such a mishap cannot occur. Gastrocolostomy should be easily avoided by positive identification of the omental attachment and taenia of the colon.

Although gastroileostomy and gastrocolostomy are rarely encountered, the incidence is undoubtedly higher than reports in the literature would indicate. Only twenty-eight cases were found in a review of the literature, but failure to recognize the condition and reluctance to bring such an error to light no doubt account for many unreported instances.

Following gastroileostomy or gastrocolostomy, the severity and type of symptoms depend upon: (1) whether a portion of the stomach has been resected, (2) whether the pylorus is patent, (3) the site of the stoma, and (4) the presence of ulceration at the stoma. The first three factors determine the amount of food short-circuited and the length of intestine functionally deactivated. Ulceration determines, to a large degree, the amount and type of pain. The usual complications of peptic ulcer, bleeding, perforation, or obstruction may occur. The most frequent complaints are diarrhea, nausea and vomiting, pain, and loss of weight.

Diarrhea usually begins soon after operation, and frequently undigested particles of food are seen in the stools. Bowel movements occur shortly after meals. Oddly enough, diarrhea is sometimes absent, and indeed constipation may occur. This has been seen in some patients in whom the stomach was not resected and the anastomosis was performed relatively high in the ileum. Undoubtedly, in such cases little if any of the gastric contents was diverted

through the stoma, but continued to leave via the pylorus, as has been shown to occur following gastroenterostomy with no pyloric obstruction.[3]

Nausea and vomiting occur in about one-half the reported cases, and the vomitus may be foul or feculent. Feculent eructations are sometimes noted, but the appetite is seldom impaired. These symptoms are due to ileal contents re-entering the stomach via the anastomosis. This has been observed roentgenographically following a barium meal and is one of the characteristic roentgenographic signs.

Abdominal pain usually does not occur unless ulceration is present. Ileal ulceration produces pain in the lower part of the abdomen and perhaps both iliac fossae. It is not as severe as pain due to jejunal ulceration and the sequence of pain—food—relief is atypical.[12, 13]

Loss of weight is usually proportional to the amount of food diverted and the length of intestine deactivated. One patient lost 92 pounds in five months. On the other hand, no significant loss of weight was recorded in several of the reported cases.

Weakness, anemia, and dependent edema are signs of malnutrition, and their presence, too, depends upon the amount of by-passed intestine.

The diagnosis usually can be made from the history alone, but it should be confirmed roentgenographically. The barium meal may be seen to leave the stomach and pass almost immediately into the colon. In some cases this cannot be demonstrated, as the entire meal may pass through the pylorus. However, the stomach may be observed to refill as the barium enters it through the ileal stoma.

It is interesting to note the interval between the performance of the gastroileostomy and its repair. The shortest time noted in the recorded cases was three months, but only about one-third of the patients were reoperated upon within a year. One patient had a gastroileostomy for twenty-five years and another for twenty-three years before they were repaired. The patients who were able to tolerate the procedure for any significant length of time were those in whom a gastroenterostomy had been performed without resection of the stomach. Upon subsequent exploration, most of these presented no serious deformity of the pylorus or duodenum.

Simple restoration of continuity may be all that is required to correct a gastroileostomy. Resection or re-resection of the stomach is sometimes necessary either at the time of repair, if feasible, or at a later date after nutritional deficits have been remedied.

Patients who have had a gastroileostomy or gastrocolostomy may present serious nutritional problems which must be corrected, in part at least, preoperatively and continued postoperatively. Vitamins, calories, and proteins are required in abundant quantities, and the electrolyte balance must be maintained. This is usually accomplished by parenteral administration of fluids, blood, and the like, but supplemental jejunostomy is a valuable adjunct in supplying the necessary food postoperatively. This has been largely overlooked in recent years since most attention has been directed to intravenous alimenta-

tion, but the procedure offers a safe and rapidly available route by which the necessary fluid, vitamins, proteins, and calories may be given. Although cramps and diarrhea may occur after administration of a formula, these are usually avoided by first offering small amounts of tap water and gradually increasing this to larger volumes. Then diluted formula or a mixture of milk and water is given. The formula is then increased in concentration and volume.

SUMMARY

Gastroileostomy is a rare surgical error, but it has no doubt occurred more frequently than the twenty-eight reported cases in the literature would indicate. One case of gastrocolostomy and two cases of gastroileostomy have been presented.

The possibility of gastroileostomy should not be overlooked either while performing gastroenteric anastomosis or while treating a patient who has had such a procedure in the past. The symptom of lienteric postcibal diarrhea in the early postoperative period is usually sufficient to make the diagnosis. The possibility should not be overlooked in patients seen many months or even years following gastric operations. Careful roentgenographic study may be necessary to visualize the lesion, and follow-up examination should be done after the barium has passed beyond the stomach. The upper portion of the jejunum should always be absolutely identified and marked before gastrojejunostomy is performed, and the level of the anastomosis should be rechecked upon completion of the operation. Adequate and often vigorous preoperative preparation is essential in these malnourished patients. Complementary jejunostomy for feeding purposes is a valuable adjunct for restoration of nutrition.

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VOLVULUS OF THE SMALL INTESTINE IN THE IMMEDIATE POSTOPERATIVE PERIOD

Report of a Case of Volvulus of the Entire Small Bowel

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VOLVULUS or torsion of the small or large intestine represents about 10 per cent of all the cases of acute intestinal obstruction seen in this country, excluding those cases resulting from strangulated external hernia.[1, 6-8] Volvulus of the small bowel accounts for 50 to 70 per cent of the total. Many European statistics vary considerably from those reported above. In these reports, volvulus accounts for from 30 to 50 per cent of all cases of acute intestinal obstruction, while small bowel volvulus represents 20 to 40 per cent of the total. The relative frequency of this condition in parts of Europe, particularly in Russia, Finland, and certain eastern European countries, is said to be due in part to the large quantity of coarse, high residue foods included in the diet in these countries. The inclusion of such foods is said to result in lengthening of the bowel, chronic distention, and abnormal peristalsis. This applies particularly to sigmoid volvulus which is especially common in these countries.

The pathogenesis of volvulus differs for each of the several types. Sigmoid volvulus is seen in those cases in which there is an abnormally large and redundant sigmoid flexure, with a long and usually narrow mesocolon. It is commonly believed that a sigmoid volvulus begins as a simple, physiologic 180 degree twist of the bowel, which is then further twisted as the bowel becomes filled with gas and stool. On the other hand, a volvulus of the right colon is always associated with an abnormally mobile cecum and failure of an adequate fixation of the mesentery. Malrotation or simple arrest of rotation of the midgut in any of the three stages described by Frazer and Robbins[2] are the underlying causes of such a condition. Such anomalies of development may also, occasionally, result in a volvulus of either the entire midgut or of the entire small intestine alone. These last two conditions are more common than was once believed. Gardner and Hart,[3] in 1934, collected eighty-eight such anomalies of intestinal rotation associated with volvulus of the entire mesentery from the world's literature. Similar cases have been seen in the Babies Hospital and Columbia Presbyterian Medical Center, and a report on twenty cases was made by McIntosh and Donovan[4] in 1939.

If there is complete nonrotation with failure of secondary fixation of the mesentery, a very long, common ileocecal mesentery associated with a short dorsal mesenteric attachment is found. Volvulus in such a case will involve the right colon as well as the small intestine. However, if partial rotation

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has taken place and the cecum has become arrested in the right upper quadrant, a narrow mesenteric pedicle from the cecum to the near-by duodenojejunal junction may permit a volvulus of the entire small intestine alone. Various degrees of malrotation and associated deficient intestinal fixation may also give rise to this condition. Volvulus of the small intestine alone accounted for approximately 27 per cent of Gardner and Hart's collected series of eighty-eight cases of volvulus due to anomalies of intestinal rotation.

In adults, however, the majority of the cases of small bowel volvulus are the direct consequence of postoperative or inflammatory adhesions and, in a smaller number of cases, congenital bands. Adhesions attached to Meckel's diverticulum are occasionally at fault and have accounted for two of the cases observed in this hospital. In other cases the bowel has become twisted about a round ligament following an operation for uterine suspension, or the bowel has become incarcerated in an internal hernia. Mesenteric cysts, tumors, intraluminal foreign bodies, and gallstones are other causes of volvulus which have been recorded in the literature.

Volvulus of the entire small intestine associated with a long mesentery but without other findings of significance, including anomalies of development, is much more unusual. McKechnie and Priestly[5] in a series of thirty-seven cases of volvulus of the small bowel, collected over a period of twenty-five years at the Mayo Clinic, have recorded only one such case. Finally, there are those uncommon cases of volvulus which have occurred within a very few days after an abdominal operation where no anomaly or predisposing causes could be found. McKechnie and Priestly mention two such cases but do not describe the extent of the twist or amount of bowel involved.

The patient whose case history is reported here belongs to this last group, and the case is reported not primarily because of its rarity (although a report of an identical case with a volvulus of similar magnitude was not found in the literature), but because it is felt that study of such a case might possibly prevent other such occurrences. Two other similar, but less extensive, cases have been found in the records of the Presbyterian Hospital in New York.

CASE REPORT

A. B. (P. H. No. 970880), a 28-year-old woman, para 0, gravida 0, was admitted to the Sloane Hospital Gynecological Service on Oct. 13, 1949, with the provisional diagnosis of an unruptured tubal pregnancy. The past history was essentially negative. She had had no serious illnesses and no previous operations. On Oct. 13, 1949, under caudal anesthesia a culdoscopy was carried out in the knee-chest position, and an unruptured left tubal pregnancy was found. She was then immediately taken to the operating room where a left salpingectomy was done under cyclopropane and nitrous oxide anesthesia. The operation was carried out without difficulty with the patient in a low Trendelenburg position. The bowel was packed off into the upper abdomen and held in place with several folded abdominal pads. During the course of the operation there was a moderate amount of straining, necessitating on several occasions the replacement and retraction of bowel and the replacement of pads.

The patient's immediate postoperative course was uneventful, and during the first day she complained only of moderate pain in the operative area. On the second postoperative day she began to complain of severe gas pains. There was some elevation of the pulse. Ambulation was carried out, and the patient spent one-half hour on the sun porch. The "gas pains"

continued throughout the rest of the day, and the abdomen was slightly distended. The pains were only slightly relieved by enemas and a rectal tube. The patient's abdominal pain continued through the third day, and she stated that she was unable to expel gas. Repeated enemas were given with only temporary relief. The pulse continued elevated.

The patient was then seen by a Surgical Consultant on the afternoon of the fourth postoperative day. By that time, she was complaining bitterly of periumbilical, crampy pain with some radiation through to the back. The pains were intermittent, occurring every ten minutes, and were associated with nausea, belching, and a sensation of gaseous distention. She had begun to vomit small amounts of fecal-smelling fluid. Although some flatus had been passed on the third postoperative day, none had been passed on the fourth day.

On physical examination the temperature was found to be 101.8° F. and the pulse 132. The patient was apprehensive and restless. The lips and tongue were dry. The abdomen was distended and tympanitic. There was a moderate degree of generalized abdominal tenderness, both direct and rebound, but this was not associated with any clear-cut muscle spasm. Peristalsis was absent. Rectal examination revealed only slight tenderness on motion of the cervix. There were no pelvic masses. Abdominal roentgenograms showed several moderately dilated small bowel loops in the left upper quadrant. There was a small amount of gas interspersed throughout the transverse, descending, and sigmoid colon. Differential diagnosis lay between that of a high small bowel, partial mechanical obstruction, and a paralytic ileus. The absence of peristalsis and the rising pulse and temperature were disquieting factors, and these together with the abdominal signs pointed at first toward a diffuse peritoneal inflammation. A Miller-Abbott tube was then passed, and the patient was started on a course of antibiotics. Electrolyte studies were carried out, and the patient was given parenteral fluid replacement.

During the succeeding forty-eight hours, the patient was followed closely by repeated roentgenograms and blood studies, and she was seen by a number of consultants. The pain and vomiting continued, and the Miller-Abbott tube drained copious amounts of foul-smelling fluid. The tip of the tube, however, despite repeated efforts, could not be passed any farther than into the descending limb of the duodenum. Electrolyte determinations were carried out at frequent intervals and fluid and electrolytes replaced parenterally. Nevertheless, in the early morning of the sixth postoperative day, the patient appeared to be in a state of collapse, with cold moist skin and an unobtainable blood pressure. Electrolyte studies done several hours before had shown a direct sodium of 127.3 meq. per liter. She had received 3,500 c.c. of fluid replacement parenterally during the day. However, by totaling the fluid deficit for the preceding twenty-four hours, it was then calculated that she had lost approximately 6,700 c.c. of fluid, of which 4,500 c.c. were by Miller-Abbott tube. New blood studies were then carried out at 3 A.M., and it was found that the patient's sodium was now 117, the potassium 3.9, and the chlorides 92. It was strikingly apparent that she was in a state of sodium deficiency shock, and the subsequent response to intravenous saline solution was dramatic. In the next twelve hours, she was given 5,600 c.c. of fluid consisting of blood, saline solution, dextrose in water, and 1/6 molar sodium lactate.

In the meantime, the patient's crampy, intermittent, upper and mid-abdominal pain continued, and it was still associated with some radiation through to the back. The abdomen remained distended and tender and without spasm or audible peristalsis. There was some evidence of shifting dullness. One loud, high-pitched peristaltic rush was heard while the lung bases were being examined. The roentgenograms continued to show a moderate distention of small bowel loops largely in the left upper quadrant (Fig. 1). The evidence for peritonitis now seemed less clear cut due to absence of sustained fever, adequate leucocytosis, and typical abdominal findings. In favor of a high, small bowel, mechanical obstruction were the continued, intermittent, crampy abdominal pains and the massive fluid loss into the gastrointestinal tract. A high obstruction would explain failure of the Miller-Abbott tube to pass and the failure to hear peristaltic sounds. A volvulus of the upper small bowel now seemed the most likely diagnosis.

The patient was then transferred to the Surgical Service, and abdominal exploration was carried out the afternoon of Oct. 21, 1949 (the sixth postoperative day), under cyclo-

propane anesthesia. The abdomen was entered through the old lower midline abdominal wound, and the abdominal cavity was found to contain approximately 1,000 c.c. of slightly turbid, straw-colored fluid which clotted on standing. The entire small bowel from just distal to the ligament of Treitz down to within eighteen inches of the ileocecal valve was found to be dilated, hypertrophied, edematous, and a dusky red color. The mesenteric vessels were greatly engorged. The small bowel in almost its entirety had been twisted on its mesentery in a counterclockwise direction a complete 360 degrees. The cecum was slightly distended, but was in its normal location, and the remaining large bowel was of normal appearance.

Fig. 1 (Case A. B.).—Roentgenogram showing moderately distended small bowel loops in the left upper quadrant. Note the virtual absence of gas shadows in either the small or the large intestine throughout the other quadrants of the abdomen.

The pelvic organs were inspected, and the site of reperitonization in the region of the left adnexa appeared to be intact. There were no intraperitoneal adhesions, and there was no apparent developmental anomaly which could help account for the occurrence of the volvulus. It was thought at the time that perhaps the small bowel mesentery was somewhat longer and more redundant than usual. After enlargement of the incision in the midline, the entire small bowel was eviscerated, and detorsion was carried out. Deflation of the small bowel was accomplished through an enterotomy in the terminal ileum. It was found possible to telescope

approximately one-half of the small bowel around the suction tip, thereby enabling the operator to remove 1,000 c.c. of intestinal contents together with a large quantity of air. The intestinal contents of the upper small bowel were then "milked" downward until they too were aspirated. The enterotomy wound was then closed with interrupted fine silk in two layers; the abdominal cavity was irrigated with sulfamylon-streptomycin solution and the wound closed. Because of a low hematocrit and low blood volume, 1,000 c.c. of whole blood were given during the course of the operative procedure.

The day after operation the patient was remarkably improved. The abdomen was soft and flat, and she stated that she was hungry. The Miller-Abbott tube continued to drain copiously and therefore was left in place. Fluid and electrolyte balance were maintained, and daily studies of the sodium, potassium, carbon dioxide, and chlorides were carried out. The patient's temperature subsided slowly. On the fourth postoperative day peristalsis became active, and on the fifth day the Miller-Abbott tube was removed. By the fifth day, also, the hematocrit, plasma protein, and electrolyte values were all within normal limits. On the seventh day the patient was having normal bowel movements and taking a low residue diet. Wound healing was uneventful, and the patient was discharged asymptomatic on the eleventh postoperative day.

DISCUSSION

This case has been presented because of its general interest, and also because it emphasizes in a striking manner what is generally thought to be only a minor factor in surgical technique; i.e., the proper replacement of bowel at the conclusion of an abdominal procedure. Yet this factor, we believe, may result in a serious, albeit a rare, postoperative complication, as in the case presented. A volvulus of the entire small bowel which is not due to adhesions, bands, or any gross developmental anomaly, occurring in the immediate postoperative course of a healthy 28-year-old woman, is certainly an uncommon event and is, therefore, of more than usual interest. A survey of all the cases of volvulus at the Presbyterian Hospital during the eighteen-year period from 1932 to 1949 inclusive has revealed only two additional similar cases (see Table I). During this period, forty-one adults with volvulus of the small and large intestine have been operated upon. An additional fifty-six patients diagnosed in the Babies Hospital during this period were not studied. The great majority of these cases were secondary to developmental anomalies of the type described at the beginning of this paper.

A brief analysis of the two cases previously encountered at the Presbyterian Hospital and a comparison between these two cases and the one just presented will allow us to formulate a theory as to the pathogenesis of volvulus in these patients.

V. W. (P. H. No. 527974), a 16-year-old girl, was admitted to the hospital with a diagnosis of appendicitis. Appendectomy was carried out on Aug. 5, 1937. A McBurney incision was used, and a Weir extension had to be made in order to expose the appendix and deliver the ileum. Early on the third postoperative day, the patient began to complain of pain and abdominal distention. This was soon followed by vomiting. On Aug. 9, 1937, the patient was operated upon.* A counterclockwise twist of the proximal ileum was found (its extent was not mentioned). Detorsion was carried out, and the patient recovered without complication.

A. L. (P. H. No. 935236), a 44-year-old man, was subjected to an abdominoperineal resection for carcinoma of the rectosigmoid on Jan. 19, 1949; the operation was car-

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TABLE I. VOLVULUS OF THE SMALL AND LARGE INTESTINE IN ADULTS, PRESBYTERIAN HOSPITAL, NEW YORK CITY, 1932-1949, INCLUSIVE

LOCATION	PATHOLOGIC FINDINGS	NUMBER	PER CENT
Sigmoid colon	Redundant flexure	10	24.4
Cecum	Mobile cecum with lack of fixation of mesentery; adhesions in 2 cases	7	17.1
Transverse colon	Redundant loop in distal half; adhesions	1	2.4
		23	56.1
Small bowel			
Duodenum	Postoperative gastroenterostomy; adhesions	1	
Terminal ileum	Cause unknown	1	
Proximal ileum	Four days postoperative appendectomy; no adhesions or anomaly found	1	
Ileum	Four days postoperative abdominoperineal resection; no adhesions or anomaly found	1	
Entire small bowel	Six days postoperative left salpingectomy; no adhesions or anomaly found	1	
Small bowel Volvulus (jejunum and/or ileum)	Secondary to adhesions, congenital bands, Meckel's diverticulum, internal hernia, and (in 1 case) a twist about a round ligament which had been used for a uterine suspension	18	
Total		41	

ried out through a lower abdominal, left paramedian incision. Early on the third postoperative day, this patient, too, began to complain of abdominal pain and distention. The colostomy was irrigated on several occasions without any improvement. By the next day (Jan. 23, 1949) signs of an acute abdominal emergency were unmistakable. At operation* a 360 degree clockwise twist of the ileum was found. The volvulus in this case had progressed to the state of gangrene; therefore, resection of five and one-half feet of the ileum was carried out. Again no apparent cause for the volvulus was found.

Study of these two cases (together with the one described earlier) will reveal several similarities. (1) In each case manipulation of the small bowel was found necessary at the initial operation. In two of the cases a Trendelenburg position was used, and the small bowel was padded off; in the third case, examination of the ileum was carried out through a McBurney incision with a Weir extention. (2) Again in each case it is to be noted that the symptoms of abdominal pain, distention, and inability to pass gas had not become severe until early on the third postoperative day. It can be postulated, therefore, that twisting of the bowel had taken place during the operative procedure and had not been corrected at that time. Failure of later spontaneous readjustment of the bowel through peristaltic action can be attributed to the paralytic ileus which occurred secondary to manipulation of the bowel during the course

*By Dr. A. F. Thompson and Dr. D. V. Habif.

of the operation. Strangulation then occurred when gaseous and fluid distention of the loops in question had progressed to such a degree that virtual locking of the bowel in the twisted position had taken place.

The roentgenograms taken in our most recent case are of particular interest and warrant further discussion. Although early in the postoperative course of this patient the roentgenographic pattern suggested to most observers a paralytic ileus as much as it did a mechanical type of obstruction, later roentgenograms (see Fig. 1) were more strongly suggestive of a mechanical upper small bowel obstruction than they were of a simple ileus. Further, although no criteria could at first be found that would lead to the diagnosis of small bowel volvulus, in retrospect, now that the operative findings are known, one important fact stands out. This fact is the presence of virtually all of the distended, gas-filled loops in the left upper abdomen. Twisting of the small bowel mesentery 360 degrees in a counterclockwise direction would tend both to shorten the mesentery and to bring the small bowel loops to lie in the left upper abdomen in the direction of the normal axis of the posterior mesenteric attachment. However, substantiation of this theory awaits further roentgenograms of other cases of volvulus of the entire small bowel.

An additional point of interest in this most recent case is the development of a striking degree of fluid and electrolyte imbalance as a direct result of the high small bowel obstruction, with its culmination in a state of sodium shock. Because of the tremendous fluid loss, both into the wall of the bowel and into the peritoneal cavity (as well as into the lumen of the bowel), it was necessary to give the patient more than 8,000 c.c. of parenteral fluids on each of two consecutive days in order to maintain homeostasis.

Finally, although we have no proof that the placement of abdominal pads and the rearrangement of the small bowel loops had any etiological relationship to the subsequent development of torsion in the last case, nevertheless, it is agreed by all those who followed the patient that this is most likely. Some straining by the patient had taken place during the procedure, and on a number of occasions, as a loop of bowel appeared in the operative field, the packs were readjusted and, with the bowel, were again pushed into the upper abdomen. At the close of the relatively simple procedure, the packs were merely removed and the wound closed without conscious effort to rearrange the bowel.

SUMMARY

1. A case history is reported of a patient with a volvulus of the entire small bowel which occurred in the immediate postoperative course following a left salpingectomy.

2. Two additional cases of small bowel volvulus occurring in the immediate postoperative period have been summarized briefly from the records of this hospital.

3. It is suggested that careful replacement of the intestines to their approximately normal location in the peritoneal cavity at the conclusion of an abdominal operation may tend to prevent the occurrence of similar cases of volvulus during the immediate postoperative period.

4. A brief résumé of the cases of small and large bowel volvulus in adults seen in the Presbyterian Hospital, from 1932 to 1949 inclusive, has been given.

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DELAYED CERVICAL ESOPHAGOESOPHAGOSTOMY WITH INTRATHORACIC TRANSPLANTATION OF THE STOMACH FOR CONGENITAL ATRESIA OF THE ESOPHAGUS WITH ESOPHAGOTRACHEAL AND ESOPHAGOBRONCHIAL FISTULA

Case Report

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CONGENITAL atresia of the esophagus with tracheo- or bronchoesophageal fistula is under ideal circumstances treated by a right-sided extrapleural approach with separation of the fistula, closure of the opening in the respiratory passage, and end-to-end anastomosis of the esophageal segments. In many cases, the gap between the proximal and distal ends of the esophagus is so great as to prevent direct anastomosis.[1-8] In at least two such cases an immediate transpleural esophagogastrostomy in one stage has been reported.[9, 10] Fatality in one case[9] was due to aspiration pneumonia following a 2-ounce feeding on the first postoperative day, and in the other[10] was due to associated cardiovascular anomalies. Swenson[6] reported a case in which immediate transpleural esophagogastrostomy for simple atresia without fistula was successful. The general practice, however, has been to close the fistula, ligate both ends of the esophagus, and later perform gastrostomy and cervical esophagostomy.[11] Re-establishment of continuity in the upper gastrointestinal tract can then await complete recovery and further development of the infant. The three most commonly used methods are: (1) the formation of an antethoracic skin tube,[4] (2) antethoracic transplantation of the stomach with esophagogastrostomy,[12, 13, 14] or (3) antethoracic transplantation of a jejunal loop.[15]

More recently the use of intrathoracic transplantation of the stomach or jejunum and esophagogastrostomy or esophagojejunostomy with the cervical esophagus have been performed successfully.[14, 16, 17]

In all reported cases of multiple stage repair which have come to our attention, a segment of distal esophagus has been present and was sacrificed. One reason for failure to use this length of distal esophagus has undoubtedly been fear for the viability of a segment of esophagus separated from its parietal vascular attachments.[18] It is a common concept that dissection of a segment of esophagus results in a nonviable esophagus, although experimental studies in dogs would not seem to support this apprehension.[19]

This report of a single case of delayed multiple stage intrathoracic repair is justified on the basis of the excellent result obtained by the use of the distal esophagus for esophagoesophagostomy in the neck, necessitating a less drastic transplantation of the stomach into the chest.

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CASE REPORT

The patient was born Nov. 20, 1947, of a 35-year-old mother, who had been a subject of long-standing bronchiectasis with previous empyema. This was a first pregnancy, complicated at the seventh month by hydramnion, which was controlled by restricted fluids, salt-free diet, and ammonium chloride. At birth the baby was resuscitated with difficulty after artificial respiration. All feedings were regurgitated and the child began on the second day to vomit a bloody, frothy mucus. No meconium was passed. Lipiodol given by mouth showed a complete obstruction of the esophagus without obvious connection with the trachea (Fig. 1, *A* and *B*). The stomach contained air indicating a distal communication with the air passages.

The child was admitted to the New Haven Hospital on Nov. 25, 1947, four and one-half days after birth. Examination revealed a small (2,410 Gm.), lethargic, slightly icteric, moderately dehydrated male with a weak cry. There was much mucus in the pharynx. There was retraction of a depressed sternum during inspiration. There was dullness to percussion over the right upper lobe and the breath sounds were obscured by rhonchi from the pharynx. Roentgenogram revealed consolidation of the right upper lobe and pneumonia of the left upper lobe and air in the gastrointestinal tract.

The first operation was performed on Nov. 27, 1947, through a posterior retropleural approach removing short segments of ribs 3, 4, 5, and 6. The proximal pouch was found firmly adherent to the trachea in the upper mediastinum but it was only after extensive dissection that the distal esophagus was found to arise from the left lower lobe bronchus. After separating the esophagus and bronchus the defect in the latter was closed with interrupted sutures of fine silk, and the esophagus was ligated. The proximal pouch was also found to communicate with the trachea by a small fissure which was closed with interrupted fine silk sutures. It was impossible to approximate the two ends of the esophagus.

On Nov. 30, 1947, a Stamm gastrostomy was performed and on Dec. 4, 1947, the blind proximal pouch was exteriorized above the clavicle on the left, through a left-sided neck approach. The patient was discharged on Jan. 11, 1948, in improved general condition. He was followed in the outpatient department, where he continued to develop normally, except for the presence of a distinct coarse horizontal nystagmus, thought to be due to a congenital anomaly.

He was readmitted on Oct. 17, 1949, at the age of 23 months, for establishment of continuity of the upper gastrointestinal tract. At this time he looked well and weighed 32 pounds. The general physical examination was essentially negative, except for the esophagostomy, the gastrostomy and the nystagmus. Roentgenograms of the stomach after injection of barium through the gastrostomy revealed a large stomach with a definite distal esophageal stump, well filled with barium (Fig. 2, *A* and *B*).

On Oct. 22, 1949, under endotracheal gas-oxygen anesthesia, the left chest was entered through the bed of the eighth rib. The distal esophagus was first dissected free, was found to be more than 6 cm. in length and of at least normal diameter for a child of this size. The diaphragm was opened and the proximal stomach mobilized by section of the left gastric artery very close to its origin from the celiac axis, the short gastric arteries, and the gastrocolic ligament down to the right gastroepiploic vessels. The gastrostomy was dismantled from within the abdomen, and the opening in the stomach was closed. Throughout this part of the procedure, the child was in constant difficulties with short periods of respiratory arrest and transient cardiac arrest on five occasions. The endotracheal tube slipped out during one manipulation, and complete cessation of respirations and cardiac arrest occurred. This persisted five minutes while the circulation was maintained by cardiac massage and the situation was relieved after an adequate airway was obtained. The operation was rapidly brought to a conclusion by bringing the stomach well up into the chest and the closed distal esophagus out through a stab wound in the anterior second intercostal space. The thoracotomy wound was closed in the usual way.

The patient's postoperative course was satisfactory. On the second postoperative day the distal esophagus was opened and a small tube was inserted into the stomach through the

Fig. 1,*A*. Fig. 1,*B*.

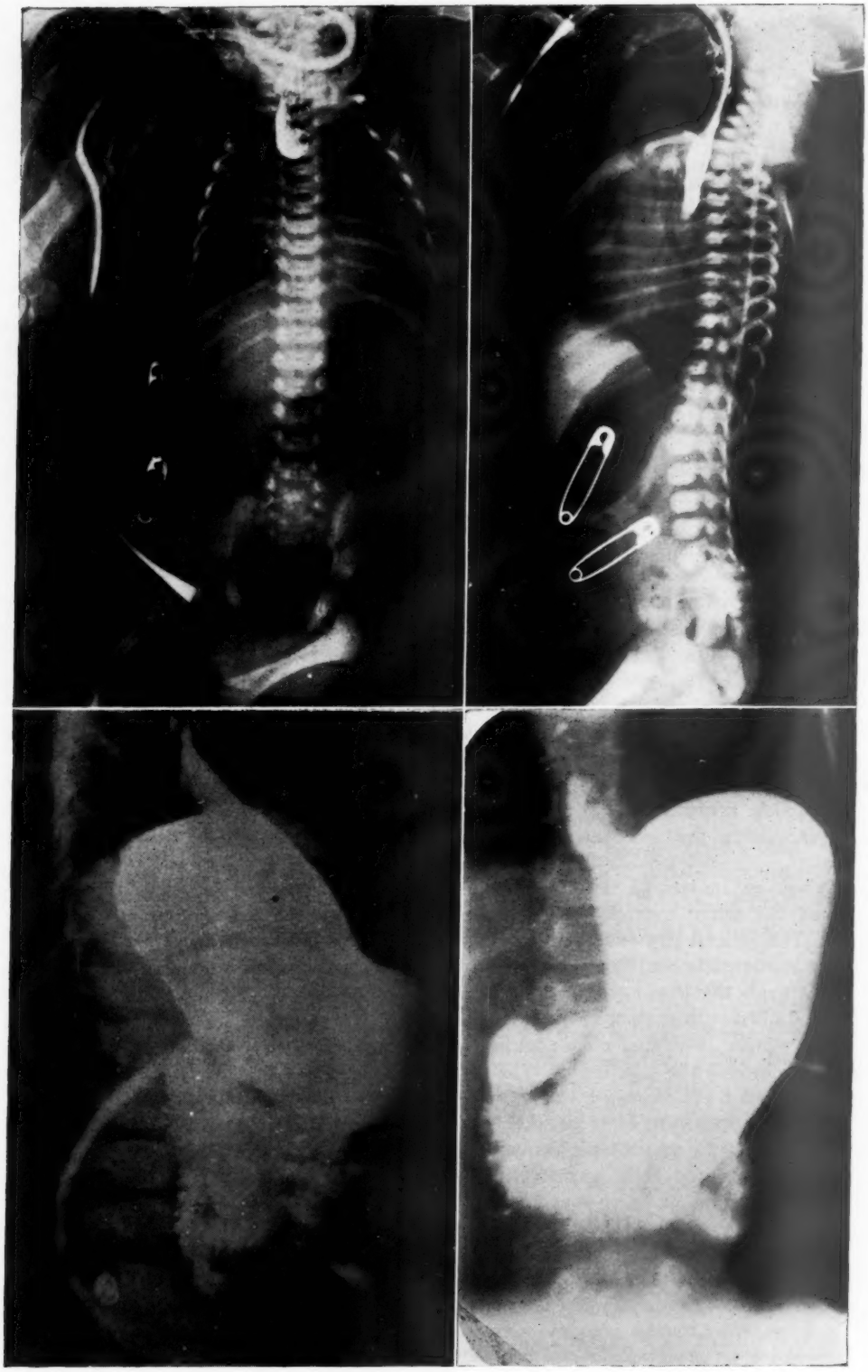
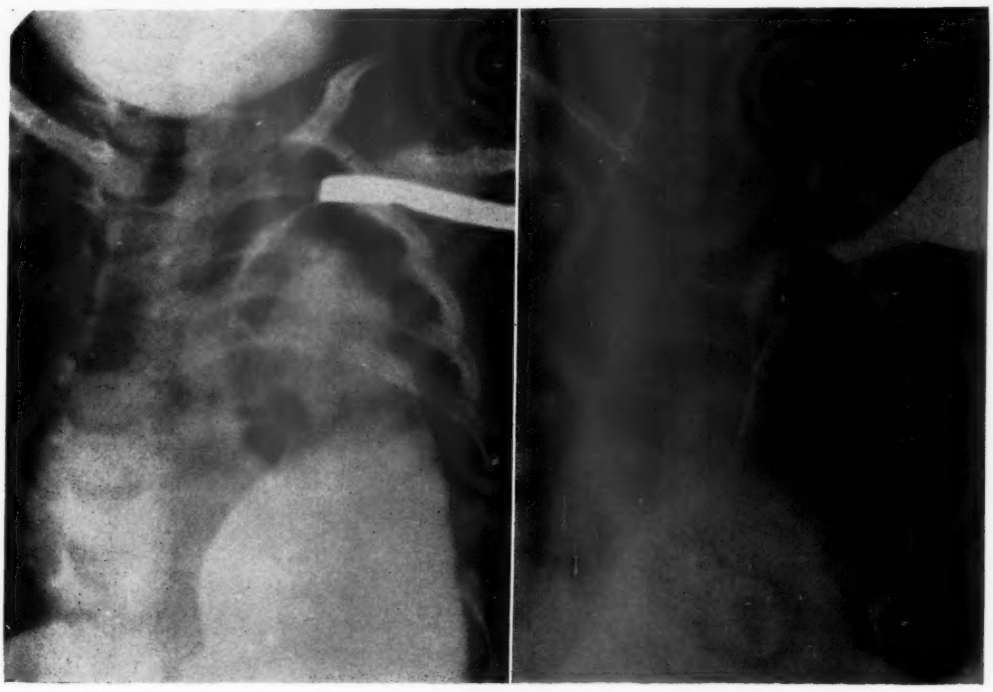


Fig. 2,*A*. Fig. 2,*B*.

Fig. 1, *A* and *B*.—Roentgenogram taken after instillation of Lipiodol to demonstrate proximal pouch. Note air-filled stomach. The bilateral pneumonia cannot be determined from these reproductions.

Fig. 2, *A* and *B*.—Roentgenogram of barium-filled stomach and distal esophageal stump. Barium instilled through gastrostomy tube. Note large size of esophageal stump.

Fig. 3.—Roentgenogram (sixth postoperative day) to show dilated intrathoracic stomach with fluid level. Some displacement of heart to the right.



A. *B.*

Fig. 4, *A* and *B*.—Filling of stomach through distal esophagostomy tube (*A*), and with syringe (*B*). The latter outlines the esophagus best.

Fig. 5.

Fig. 6.

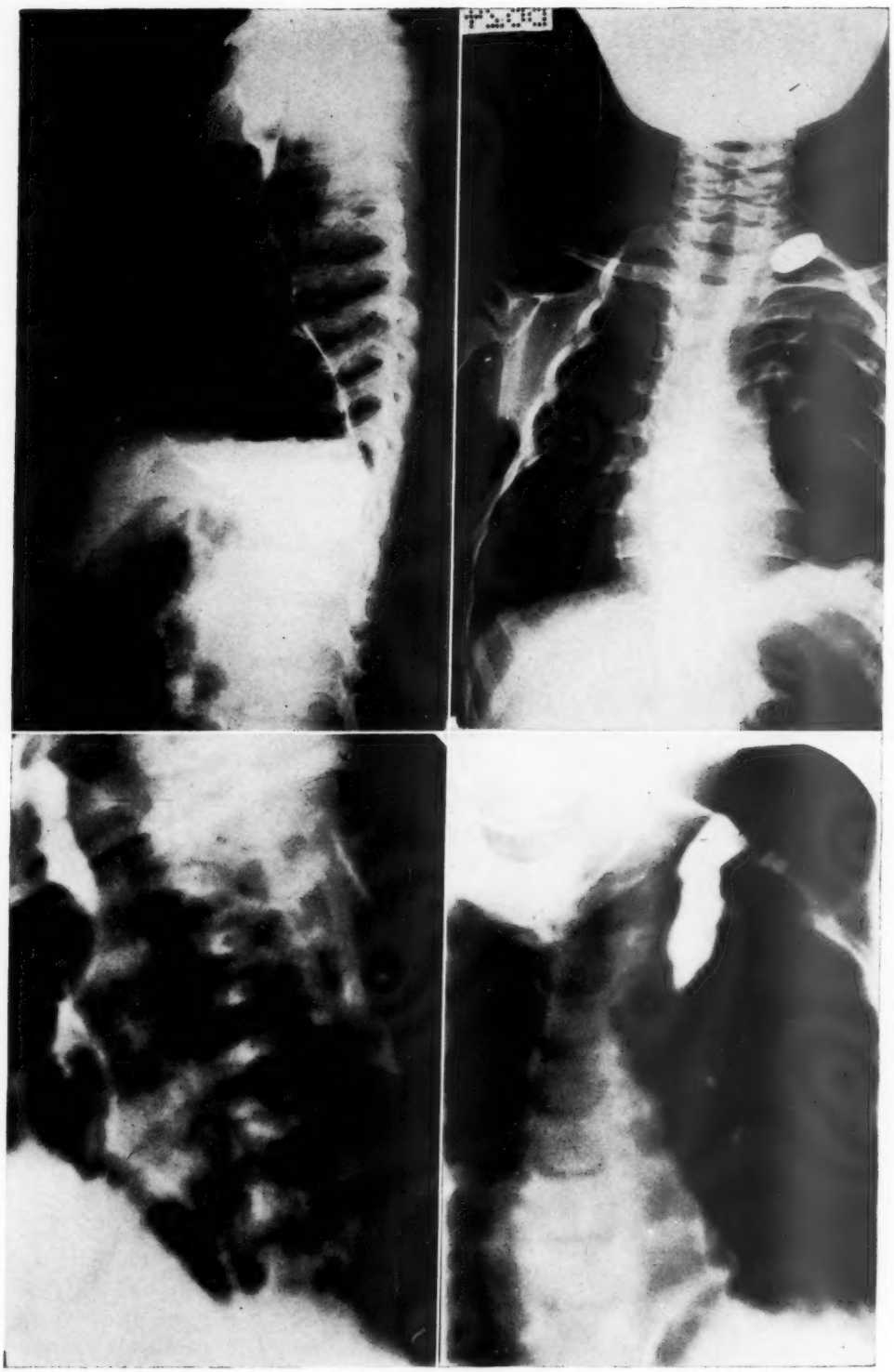


Fig. 7,*A*.

Fig. 7,*B*.

Fig. 5.—Roentgenogram of barium swallow twenty-eight days after esophagoesophagostomy. Good function apparent despite angulation and filling defects.

Fig. 6.—Roentgenogram before removal of pennies. Hold-up is actually below point of anastomosis at about level of pleural opening. Note decrease in size of intrathoracic stomach and shift of heart back toward the left.

Fig. 7, *A* and *B*.—Barium swallow ten months after final stage. Note angulation still present but with improvement in function.

esophagus. Feedings in small amounts, 20 c.c. per hour, were started on the third postoperative day and rapidly increased to a normal liquid diet for a 23-month-old child. Roentgenograms showed dilatation of the stomach with an air fluid level (Fig. 3).

A barium study on the sixteenth postoperative day revealed that the esophagus coursed medially to the distended stomach and entered the stomach at the level of the sixth rib (Fig. 4, *A* and *B*). Retention, as measured by repeated aspiration of the stomach and by x-ray examination, indicated a very slow emptying of the stomach, probably the result of vagus section.

Fig. 8.—Patient on Jan. 12, 1951, at about 38 months of age. The slight protuberance of the esophagus in the neck can be seen as well as the anterior scars of operation.

On Nov. 12, 1949, after lavage of the stomach, the final stage, a cervical esophagoesophagostomy was performed. A Z incision starting in the neck just above the sternoclavicular joint was carried laterally around the cervical esophagostomy, then the middle limb was carried downward and medially to the medial end of the incision in the second intercostal space, and thence laterally around the distal esophagostomy and to the anterior axillary line for the lower transverse limb. Cuffs of skin were used to close both ends of the esophagus. The proximal esophagus was freed up for a distance of 4 to 5 cm., down to the scalene muscles.

A flap of pectoralis major muscle was turned laterally and inferiorly from its origin to the sternum and clavicle, and the pleural cavity was entered through the second intercostal space. The distal esophagus was dissected free from these structures. The neck dissection was then carried down over the scalenus muscles to the dome of the pleura. A Kelly clamp was pushed up against the dome of the pleura from within, the fascia and the pleura

were incised, and then a clamp in reverse direction was passed through, and the distal esophagus drawn up into the neck. The intercostal incision was then closed with silk about a small catheter for drainage of the pleural cavity.

The two ends of the esophagus could be overlapped 4 cm. without tension. The ends of esophagus were trimmed and an end-to-end anastomosis was made in three layers with a running lockstitch of catgut in the mucosa, mattress sutures of silk in the muscularis and submucosa, and interrupted sutures of silk in the outer muscle layer. Before the anterior surface was closed, the catheter in the proximal pouch was passed through the anastomosis and left in place. The pectoralis muscle was resutured to its origin and closure of the wounds carried out in the usual way.

The patient's postoperative course was relatively uneventful. The esophageal catheter was inadvertently removed on the day of operation and was reinserted into the upper esophagus, above the anastomosis. The highest temperature was 101.5° F. on the first postoperative day. The pleural catheter was removed on the fourth postoperative day. Fluids by mouth were started on the fifth postoperative day, and the diet was increased to include puréed foods at the time of discharge on the eleventh postoperative day (Nov. 23, 1948). He was taking solid foods by Dec. 1, 1949. He had rare episodes of difficulty with swallowing large food particles but with careful massage by the mother the material could be passed through the anastomosis. On Dec. 8, 1949, a barium swallow indicated narrowing below the anastomosis (Fig. 5). Esophagoscopy on Dec. 12, 1949, revealed no evidence of scar stricture and a No. 20 French dilator was passed with ease. It was the feeling of the esophagoscopist that the obstructive symptoms were due to angulation rather than actual narrowing.

Instances of difficulty in swallowing became less frequent and after February all food could be washed down by a swallow of milk. After mid-April there was no further difficulty except for one episode in September when a grape became stuck at the point of angulation and again on Oct. 4, 1950, when he attempted to get two pennies past the area of angulation (Fig. 6). These had to be removed using the child size anterior commissure laryngoscope.

A roentgenogram on Sept. 13, 1950, still showed angulation, slight narrowing without delay at the anastomotic site, and gastric dilation (Fig. 7, *A* and *B*). Since that time there has been no further difficulty (Fig. 8).

Comment.—Except for the anesthesia complications, the entire procedure could have been performed in a single stage. However, control of the gastric dilatation was probably easier through the esophagostomy and in addition the viability of the transplanted distal esophagus could be determined with much less danger than with an anastomosis lying deep in the neck in direct continuity with the chest cavity and the soft tissues of the neck and mediastinum.

There was some question as to the type of procedure to be carried out at the time of the second stage. Two possibilities in addition to the procedure performed were seriously considered: (1) an anastomosis beneath the skin over the clavicle, and (2) resection of a segment of clavicle and first rib with the anastomosis then being made extrapleurally beneath these bones. The first was ruled out because of the fear of obstruction due to the angulation at the chest wall and over the clavicle, and the danger of injury to the esophagus on bony protuberances, when protected only by skin and subcutaneous tissues. The second was decided against on the basis of the danger of compression by scar tissue or regenerating bone.

SUMMARY

A case of congenital atresia of the esophagus with proximal esophagotracheal fistula and distal left lower lobe bronchoesophageal fistula treated by

multiple stage procedures with eventual cervical esophagoesophagostomy has been reported.

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RADICAL PANCREATODUODENECTOMY WITH RÉSECTION AND REANASTOMOSIS OF THE SUPERIOR MESENTERIC VEIN

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THE biology of cancer of the pancreas is characterized by extreme malignancy and the rapacious growth of its metastases. The surgical therapy of this disease has been ineffective. Only one patient undergoing radical pancreatoduodenectomy for a carcinoma of the pancreas at this clinic has survived five years. This patient, originally reported by Dennis and Varco,[1] has now survived six years.

This case report concerns a supplementary and futile attempt to remove a carcinoma of the pancreas completely by resecting a segment of the superior mesenteric vein encroached by the tumor.

Mrs. S. A., a 76-year-old woman, was admitted to the University of Minnesota Hospital with a three-week history of jaundice, anorexia, and vague epigastric pains. The patient stated that she had lost 20 pounds in weight during the three weeks previous to admission. She had enjoyed excellent health until nine months previous to her admission, at which time she complained of polyuria, polyphagia, and polydipsia. Her personal physician had diagnosed the trouble as diabetes and initiated insulin therapy (30 units, regular, and 10 units, protamine).

Examination revealed an emaciated (90 pounds), markedly jaundiced, elderly white woman whose mental facilities were excellent. Abdominal palpation revealed a huge distended gall bladder reaching the level of the umbilicus. The liver itself was tender and extended 8 cm. below the costal margin. A deeply-situated, fixed mass could be felt in the area of the pancreas. No ascites was evident. Rectal examination was negative.

Pertinent laboratory reports were: hemoglobin 10.9; blood glucose (fasting) 148; total calcium 7.8; serum bilirubin 1 minute, 15.4 mg. per cent, total 25.1; alkaline phosphatase 17.4 Bodansky Units; thymol turbidity 3 units; prothrombin time, patient 16.9 seconds, control, 12.1 seconds; and normal bleeding and clotting times.

A tentative diagnosis of carcinoma of the pancreas was made and the patient was transferred to a surgery ward for operative preparation.

An exploratory laparotomy May 25, 1950, was done through a transverse incision above the umbilicus. Inspection of the abdomen disclosed a 5 cm. mass in the head of the pancreas. There was no evidence of distant metastases. The regional lymph nodes were slightly enlarged, but soft in consistency. No palpable masses were present in the liver. The superior mesenteric vessels and the portal veins were not thought to be involved.

Dissection was begun by transection of the stomach through its mid-portion and removal of the gastrohepatic omentum. The hepatic artery was isolated and the gastroduodenal artery ligated at its origin from the hepatic artery. Next, the common duct was freed from enveloping tissue, clamped, and sectioned just at the junction with the cystic duct. After mobilizing the duodenum and sectioning it between clamps at the ligament of Treitz, the pancreas was cut across approximately 4 cm. to the left of the mesenteric vessels. However, further dissection revealed the tumor to be firmly attached to the lateral wall of the superior mesenteric

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vein for a distance of 2 cm. At this point it was decided to remove the vein wall involved with tumor with the hope that the defect could be resutured without performing an actual resection of the vein.

The Blalock clamps were applied to the vein on each side of the cancer plaque and the involved portion removed. It was soon evident that the resulting defect could not be resutured without leaving a residual lumen of only a few millimeters; therefore, a complete resection (3 cm.) of the area was done.

The Blalock clamps were re-applied to allow sufficient cuff for anastomosis; the proximal clamp was placed immediately below the entrance of the splenic vein. Simultaneously a clamp was also placed on the superior mesenteric artery to obviate engorgement of the intestine with blood.

After freeing up the distal segment of the superior mesenteric vein by ligating small side branches, the vein ends were sutured end-to-end with continuous everting 000000 silk sutures. The anastomosis was accomplished without event. Upon release of the clamps on the vein and artery, a normal blood flow ensued. The bowel had a reasonably normal color and consistency throughout the ninety-five minute interval between the initial application of clamps to the superior mesenteric vein for the abortive attempt to repair the vein and their subsequent removal after completion of the definitive anastomosis.

The operation was concluded by anastomosis (end-to-side) of the common bile duct to a proximal loop of jejunum, direct anastomosis of the pancreatic duct to the jejunum, and finally a closed anastomosis of the residual gastric pouch to the jejunum just distal to the previously described anastomoses.

Pathologic Examination.—The surgical specimen was a 6 cm. tumor which was present in the head of the pancreas with extension into the occlusion of the common duct. The lines of resection through the pancreas and common duct were free of cancer by microscopic examination. Metastatic tumor was found in three lymph nodes along the second portion of the duodenum. All other nodes included with the surgical specimen were negative.

Microscopically, the tumor was a poorly differentiated adenocarcinoma with moderate pleomorphism and occasional mitotic figures.

Postoperative Course.—The patient enjoyed an uneventful postoperative course and was discharged on the tenth postoperative day. The only complaints were of unusually bulky stools and a lessened appetite.

The patient returned to Clinic on the eleventh postoperative day, at which time she had no particular complaint. Jaundice had cleared and the wound was well healed. The diabetes was controlled with 15 units of regular insulin taken three times daily when urine was 3 or 4 plus for sugar.

Subsequently she failed several clinical appointments, but returned to the Surgery Out Patient Clinic forty-two days after her discharge. At this time she was cachectic, dehydrated, and complained of loss of appetite. No history of the amounts of insulin she had been taking could be obtained. Emergency chemistry tests were essentially normal. Parenteral fluids were initiated and the insulin therapy re-evaluated. In spite of these measures she continued to have an elevated temperature. soon became comatose, and died forty-eight hours after admission.

Autopsy Findings.—Ascites and numerous implants on the visceral and parietal peritoneum were present. The liver was enlarged (2,000 grams) and was at least 60 per cent replaced with tumor tissue. The anastomoses of the common duct and the pancreatic duct to the jejunum were intact but gross tumor was present at both sites. Large amounts of metastatic tumor were present in lymph nodes along the aorta, inferior vena cava, hilus of the right kidney, and along the greater curvature of the stomach.

The anastomosis of the superior mesenteric vein was nicely healed with only a slight reduction in the vein diameter (circumference—2.0 cm.). The intimal surface was smooth with no evidence of thrombus formation.

The immediate cause of death was large pulmonary emboli which apparently came from several tumor- and thrombus-filled radicles of the hepatic vein.

DISCUSSION

To our knowledge no previous resection and anastomosis of the superior mesenteric vein has been reported in the literature. In this instance both the superior mesenteric artery and vein were clamped for ninety-five minutes while the remaining dissection, segmental removal of the vein, and the subsequent anastomosis were performed. No anticoagulants were given. In this regard it should be pointed out that the patient had evidence of severe liver damage with a slightly elevated prothrombin time. The resulting defect (not proved) in the clotting mechanism probably helped to insure the successful clamping of the mesenteric vessels. It is doubtful that the vessels could be routinely clamped as long as two hours without danger in patients with a normal blood clotting mechanism.

Recently, Nelson and Kremen[4] have reported a series of experiments relating to this problem. They found that, in the dog, either the superior mesenteric artery or vein could be safely occluded for two hours. When either was clamped for at least four hours, death ensued in a majority of the animals. The occlusion of the superior mesenteric vein alone was definitely more lethal.

Preoperative administration of heparin significantly extended the period during which the vessels could be occluded. Heparin was especially effective with occlusion of the superior mesenteric vein.

In an earlier study, Martin, Laufman, and Tuell[3] found that heparin and Dicumarol did not prevent sludge formation in the smaller mesenteric vessels after occlusion of the superior mesenteric vessels, but did effectively retard thrombus formation.

A second factor influencing survival after experimental occulsion of the intestinal blood supply is the use of antibiotics which reduce the intraluminal bacterial flora. Since many investigators have substantiated this point, it will not be reviewed here. As a result of recent studies, the coagulant effects of such compounds must be considered.

The enthusiastic investigations of an infinite number and variety of blood vessel anastomoses in the last few years allow one to choose one of several techniques with which to repair a defect in either the mesenteric artery or vein. Probably the best technique is to re-anastomose the vessels if possible. Other alternatives are the anastomosis of the artery and vein to the aorta and inferior vena cava, respectively. Transplants of either autografts or homografts of veins or arteries could also be considered.

It may be possible that future studies will indicate the feasibility of deliberate sacrifice of the superior mesenteric vessels and end-to-side anastomosis to the aorta and portal vein, respectively, in those patients with a carcinomatous extension to the celiac axis region which would otherwise preclude the attainment of a "good cancer operation."[2] Unfortunately, the proximity of the vena cava, aorta, and hepatic artery; the marked physiologic problems resulting from total pancreatectomy; and the tumor's extremely rapid autonomous growth all tend to militate against further extension of the conventional pancreatoduodenectomy for carcinoma of the pancreas.

In this regard, this case history again emphasizes the necessary consideration of the biology of the carcinoma in question. This patient undoubtedly had widespread microscopic involvement at the time of operation. That is, the microscopic involvement was far in advance of the gross involvement.

This is in contrast to the experience with other tumors, such as carcinoma of the colon, in which the microscopic spread of the tumor more closely parallels the extent of the gross evidences of spread. A tumor of this sort, having a more favorable biology, would lend itself much more favorably to increasingly radical resections and the effectiveness of planned subsequent operations ("second look exploration").[5]

SUMMARY

1. A case of carcinoma of the pancreas which was treated by radical pancreatoduodenectomy and its subsequent course is reviewed.

2. In an attempt to completely remove the gross tumor a 3 cm. segment of the superior mesenteric vein was removed and the vein re-anastomosed.

3. The efficacy of utilizing anticoagulants to prevent thrombosis during and following surgery of the mesenteric veins is discussed.

4. The relationship between the biology of a carcinoma and the rationale of extending operative therapy for it is mentioned.

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CARCINOMA OF THE STOMACH

A Report of an Epidermoid Carcinoma of the Pyloric Antrum

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CANCER of the stomach is a timely topic at any time, particularly when considering such a clinical rarity as an epidermoid carcinoma of the pyloric antrum. Carcinoma of the stomach is the most lethal, if not the most frequent, visceral cancer in human beings, with a yearly toll of about 50,000 deaths in the United States. This exaggerated death rate results from the lack of preventive measures, the tardiness in establishing the proper diagnosis, and the failure to encompass the disease process in an ample and radical operation in the greatest majority of cases; it is largely due to the insidious nature of the disease, its rapid growth with a widespread infiltration of the organ, its diffuse metastasis, as well as to the relative difficulty in examining this viscus, the utter neglect of the patient in submitting himself to the proper clinical studies in due time, and to the most regrettable underestimation or unrecognition of the disease by the physician who first sees the case.

Carcinoma of the stomach is by all odds adenomatous in type (78 per cent). Only 2 per cent of the cases are scirrhous and 20 per cent mixed type. The epidermoid carcinomas are indeed rare. In the most recent review of the literature on this subject, O'Brien and Meehan[7] reported 13 adenocanthomas and only 9 pure epitheliomas of the stomach. To these I want to add another epidermoid carcinoma of the pyloric antrum.

The reason for the development of these epidermoid cancers in the stomach is not at all clear, and the hypotheses that have been advanced in their support are not generally accepted. Most authors reporting cases of this type have not dealt at any length with this fundamental consideration. Those who have given any regard at all to this topic have dismissed it by considering these tumors merely as heterotopic or metaplastic in nature. When the tumors occur in the upper segment of the stomach, this assumption may be valid, for the growth may develop from an ectopic islet of esophagic mucosa or even by continuity of an epidermoid carcinoma of the lower end of the esophagus. However, this is not acceptable when considering a squamous-cell carcinoma in the pyloric antrum. It is true, nevertheless, that epidermoid carcinomas have occurred even "in the gall bladder, which is one of the last places to expect squamous epithelium in the human body," according to Walters, Gray, and Priestley.[4]

In a review of the literature at hand on this particular subject, the only author I have found trying "to crack the nut" of the etiology of these epidermoid carcinomas in the stomach and intestinal mucosae is Duran-Jorda, now in Manchester, England. In a personal communication, dated April 13,

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1950, he stated (my translation), "Without question, it is my point of view that the adenoacanthomas, or carcinomas of stratified squamous epithelium (dermoids), originate from the flat semisquamous layer which covers and protects all the mucous membranes in the body. . . .To assume that glandular cells reverse to a flat squamous type of cell by a process of metaplasia is asking too much of our imagination. It is much simpler to show that all mucous surfaces, the glandular inclusive, communicating with the outside, have this semisquamous layer to protect them from autodigestion (stomach) and infection."

Fig. 1.

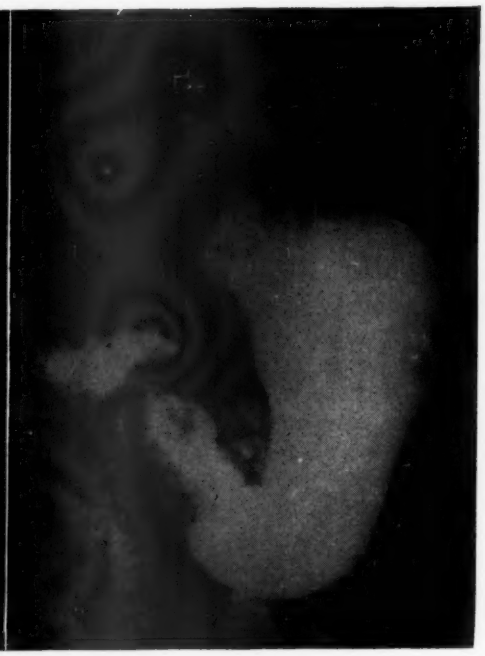

Fig. 2.

In a paper read before the Manchester Medical Society,[5] Duran-Jorda said, "The existence of a flat epithelial protective layer at the top of the mucosa layer of the gastric and intestinal mucosae will open up a new understanding of the problem of gastric and intestinal pathology and some processes which have puzzled surgeons, but which now have a simple explanation. . . . After studying the gastric and intestinal mucosae by a new process of fixation, the author describes a flat epithelial layer at the top of the gastric and intestinal mucosae. This layer is very fragile and disappears if roughly handled, or if fixed in formol saline. . . . It also disappears from post-mortem specimens. . . . The layer is formed by flat epithelial cells and contains a large quantity of diapedesic cells and a network of capillaries. . . . The existence of the layer has been confirmed in different mammals, and can also be studied in the human foetus. . . . The author considers its physiology to be connected with the protection of the mucosae from auto-

digestion by gastric and intestinal enzymes. . . . The author also describes the histologic picture as seen in the stomach, of preulcerative processes, the healing process of gastric ulcer, and postulcerative processes, and thinks that the further study of this new layer will open up a different understanding of the pathologic processes in the gastric and intestinal mucosae.''

Whether we accept the existence of this semisquamous, flat, epithelial layer overlying all mucosae of the body or not, this interesting consideration brought forth by Duran-Jorda certainly provides a more logical and scientific reason for the development of the rare squamous-cell carcinomas in the stomach, gall bladder, intestines, and other viscera lined by glandular epithelium. In view of these findings, therefore, we must not conclude that the occurrence of an epidermoid carcinoma in any of the glandular epithelia of the body is a chance occurrence on the basis of a metaplasia or of an ectopic implantation of squamous cells. It would certainly be a natural development in an existing, although elusive, ''semisquamous, flat epithelial layer lining and protecting all mucous surfaces of the body connecting with the outside,'' as described by Duran-Jorda.

CASE REPORT

N. S. R. (Hospital No. 10,116, Oncologic Hospital, Puerto Rico League Against Cancer), a thin, white, married, 73-year-old man, was admitted for treatment on Nov. 3, 1949. Past history revealed he had had measles, smallpox, and mumps. There was no history of syphilis, diabetes, tuberculosis, pernicious anemia, or tropical sprue, nor of previous operation or trauma. There was no family history of pernicious anemia, sprue, leucemia, or cancer. His chief complaint was that of a ''dyspepsia'' progressively increasing in intensity during the last few months, accompanied by anorexia and constipation in the last weeks. He had not had bloody or black stools but lately had had a coffee-ground vomitus. In the last few weeks he had lost about 8 pounds in body weight. He had followed a self-prescribed diet of low protein content and of a variable caloric value.

On physical examination the patient appeared to be an elderly, emaciated man with an adentia and a moderate peripheral arteriosclerosis. Blood pressure was 120/71, pulse 74, and respirations 20. The abdomen was soft, scaphoid, and presented a small, palpable, discrete, hard mass in the epigastrium. The liver was considered normal. There was no evidence of ascites or of portal obstruction. There was no detectable Blumer's shelf or Virchow's node.

The laboratory work-up revealed a normocytic, hypochromic anemia (red blood cell count was 3,010,000 with 64 per cent hemoglobin [8.7 Gm.]), absence of free acid and a low total acidity even after histamine stimulation, urine of low density (1.002) with traces of albumin, and a blood chemistry within normal limits.

TABLE I.

	FREE ACID	TOTAL ACIDITY
Residue (fasting)	0	16
After ingestion of 50 c.c. 7.5 per cent alcohol		
20 minutes	0	18
40 minutes	0	20
After histamine injection		
20 minutes	0	20

Roentgenologic study of the stomach, done elsewhere, showed the presence of a well-defined obstruction at the pylorus, probably neoplastic in nature, extending to the duodenal bulb (Figs. 1 and 2).

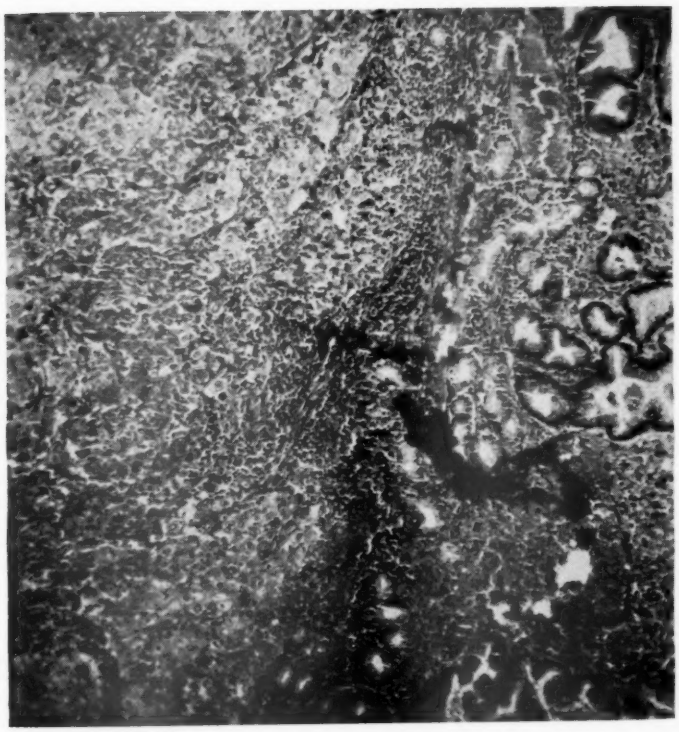

Fig. 3.

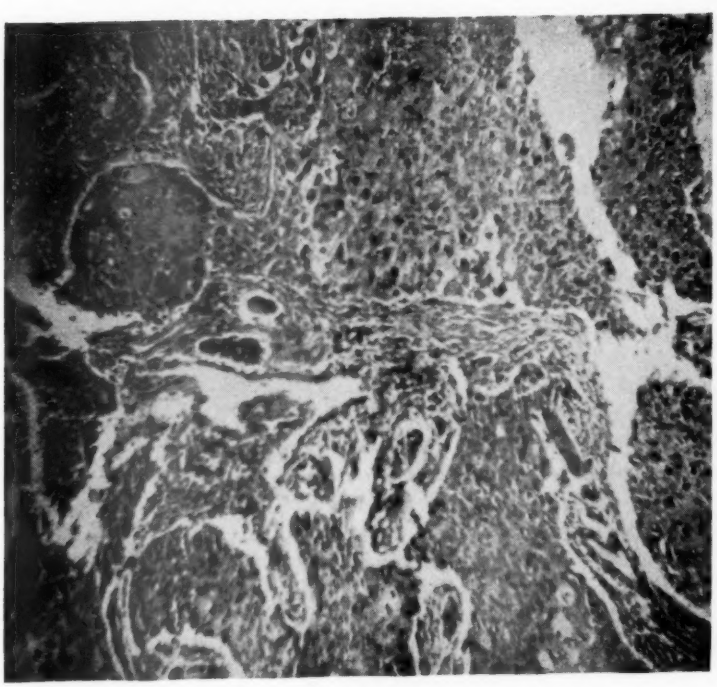

Fig. 4.

With a preoperative diagnosis of carcinoma of the pylorus, a laparotomy was performed on Nov. 23, 1949. At the antropyloric area, including the first portion of the duodenum and extending to the body of the stomach, a hard, lobular mass measuring about 5 by 5 by 6 cm. in principal diameters was found. There were no nodules found in the liver or in the paragastric ligaments. A partial resection of the stomach was done by the Hofmeister technique.

Pathologic Report.—The pathologic report* of the specimen was as follows (my translation):

Macroscopic: The specimen consisted of a segment of stomach measuring (after fixation in 10 per cent phenol solution) 12 by 7.5 cm. in principal diameters. The external surface was covered by a semitranslucent serosa somewhat thickened. The gastric part of the specimen was divided by two well-defined furrows into three lobules. The cut section corresponded to the pyloric and antropyloric areas. The lumen was almost obliterated by a huge thickening of the mucosa, which had been largely replaced by a tumor which permeated the full thickness of the wall up to the serosa. The tumor was made up of a soft, whitish, granulomatous tissue. In certain zones the mucosa was necrotic. The tumor extended by infiltration to about 7 cm. beyond the pyloric ring.

Fig. 5.

Microscopic: The stomach coats were extensively infiltrated by plexiform islets and cords made up of polygonal elements of neutrophilic cytoplasm and oval nuclei. In various zones there were well-established keratinic changes and in others connecting bridges typical of malpighian cells were observed. The stroma was lax and was densely infiltrated with monocytes. The mucosa had been replaced in various parts and the membrana propria was dense and diffusely infiltrated with round cells. The tumor extended to the pyloric ring and replaced the antral mucosa. Diagnosis was squamous-cell epithelioma, grade 3, of the stomach.

This tumor may have had its origin from the esophagus or from an ectopic area of esophagic mucosa in the stomach (Figs. 3 and 4).

The patient made an uneventful recovery and was discharged from the hospital on the seventh postoperative day under a dietary regime. Fifteen days after discharge he excoriated

*By Dr. J. E. Taveras, pathologist.

the scar and developed a huge pyogenic abscess which required ample drainage and daily irrigation with liquid Furacin and the administration of 400,000 units RR penicillin daily for ten days. This healed nicely by second intention.

The patient has been seen several times at our follow-up clinic for the last few months. He has gained weight, has no gastrointestinal tract symptoms, and shows no evidence of recurrences. On Feb. 14, 1950, a check-up study of the gastric motility was ordered. The roentgenologic report* was as follows: The radioscopic and radiographic study with an opaque meal, 83 days after partial gastrectomy, revealed a relatively normal function of the remaining gastric segment (Fig. 5).

CONCLUSIONS

Although the etiology of squamous-cell carcinoma arising in glandular epithelia is a highly controversial and largely hypothetical point, I feel that a scientific explanation of this phenomenon has been unraveled by the discovery and description by Duran-Jorda of a flat epithelial layer lining these glandular surfaces. They cannot then be considered as a chance occurrence on the basis of metaplasia or an ectopic implantation of squamous cells.

O'Brien and Meehan recently reviewed the literature on this subject and found only nine pure squamous-cell carcinomas of the stomach reported. To these I add another pathologically authenticated case of pure epidermoid cancer of the pyloric antrum.

SUMMARY

1. An epidermoid carcinoma of the pyloric antrum is presented, thus raising the total number of such cases reported to ten.

2. Consideration is given to the etiology of the lesion. A brief presentation is made of the findings of Duran-Jorda and their bearing on this problem.

Acknowledgment.—Many thanks are due to Dr. J. E. Taveras, our pathologist, for his painstaking study of the histopathology of the tumor, to Dr. Hamlet Hazim, our resident surgeon, for his cooperation in the management of the case, and to Mr. José J. Muntaner, our technician, for the roentgenograms and photomicrographs accompanying this work. My gratitude is also herewith acknowledged to Dr. I. González Martínez, our director, for his stimulus in getting this paper published, and to Dr. P. Malaret, our subdirector, for reviewing the manuscript.

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*By Dr. I. González Martínez.

CASE OF EPIGNATHUS

Survival of Host After Its Excision

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TERATOID parasites of the mouth are rare examples of what are considered to be asymmetrical double monsters. Some minor confusion has existed because of the different terminologies applied to these oral parasites. Present nomenclature has the parasite taking its name from its point of origin, that is, from the jaw, epignathus; from the sphenoid, episphenoideus; and from the palate, epipalatus or epuranus.[1] Other names, such as sphenopagus parasiticus and gnathopagus parasiticus, are obviously interchangeable with the former terms.

The stage of development of the parasite has led to other classifications. Arnold[2] divided the oral parasites into 4 groups, group 1 consisting of pedunculated, pear-shaped dermoid tumors attached to the palate or epipharynx, and the classification then proceeding through 3 more and more highly developed groups to a Group 4 consisting of monsters with recognizable extremities, eyes, and other organs. Schwalbe[3] also divided them into 4 groups, his Group 1 corresponding to Arnold's Group 4.

The recorded cases in the literature show that most of the highly developed oral parasites occur in stillborn or newborn infants, the hairy polyps and dermoid tumors in children and adults. The reason for this seems obvious. Only the small and undifferentiated parasites are innocuous enough to permit development and survival of the host. For some unexplainable reason the abnormality seems to occur more often in females.

The case to be reported represents an epignathus or gnathopagus parasiticus in a female infant; the parasite contained well-developed and easily recognizable organs, that is, umbilical cord, intestine, and parts of the bony skeleton. To our knowledge no case of survival of the host after surgical removal of an epignathus has been previously reported. The favorable outcome in this case undoubtedly was due to the prompt measures taken soon after birth, together with the fact that the oral monster was relatively small, originated in the lower part of the jaw, and was not attached to the mandible.

REPORT OF CASE

A white female infant, aged 4½ hours, was admitted to Foundation Hospital for treatment of a large deformity of the tongue which appeared to interfere with respiration. She had been delivered at term of a 19-year-old woman para i, gravida i. Except for a third degree perineal laceration sustained by the mother, delivery was uneventful. The infant cried immediately.

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Weight recorded on admission was 8 pounds, 8 ounces, temperature 98.6° F., pulse 140 beats a minute, and respiration 40 a minute. Examination revealed a newborn white female infant in acute respiratory distress, with costal and supramanubrial retraction (Fig. 1). Moderate cyanosis was present; the skin was otherwise clear, with good turgor. The fontanels were open and soft. The eyes and ears were normal. The mouth was filled with a tremendous tumor measuring approximately 12 by 7 by 7 cm.; overlying and intimately adherent to it was an extremely hypertrophied tongue. The tumor felt cystic in some areas and bony hard in others. The lips completely encircled the tumor and were applied to it so that no opening into the mouth was possible. The tumor appeared to be relatively mobile, without fixation to bony structures. The thorax was symmetrical and mobile; respirations were rapid and grunting with severe intercostal retraction. The

Fig. 1.—Photograph of patient showing epignathus in situ (tracheotomy and gastrostomy have been done).

lungs were clear to percussion and auscultation; the heart rate was 140 beats a minute without murmurs. Examination of the abdomen revealed no abnormalities. External genitals were those of a normal female infant. On rectal examination, a small membrane was noted; it was perforated by the examining finger, with subsequent passage of a large meconium stool. Extremities and neuromuscular system were essentially normal.

Because the tumor obviously obstructed the airway, tracheotomy was done 5 hours and 25 minutes after birth under 1 per cent procaine anesthesia. The incision had to be transverse, in as much as the neck was too short to accommodate the usual longitudinal incision for tracheotomy. The diameter of the trachea was so narrow (approximately 0.25 cm.) that only the inner lining tube of a Jackson number 0 tracheotomy tube could be inserted. Because the cervical structures compressed a rubber catheter, the more rigid

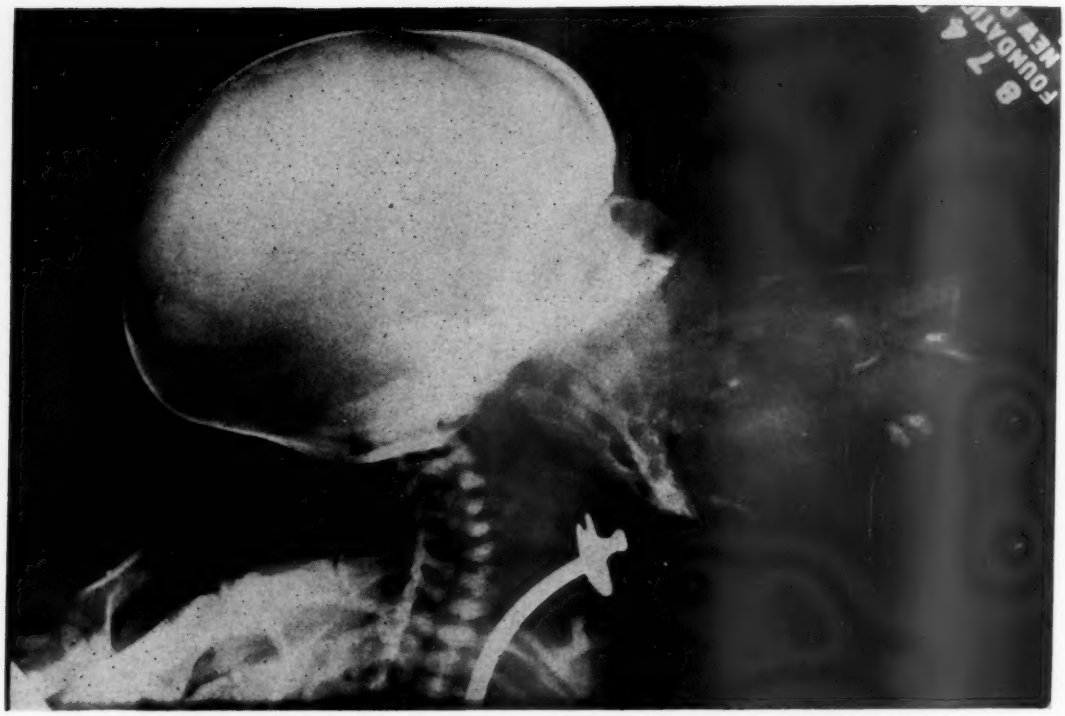

Fig. 2.—Roentgenogram showing contained long bones, teeth, and partially formed pelvic bones in epignathus.

Fig. 3.—Epignathus following surgical removal.

metal tube had to be used. There was immediate improvement in the patient's color; the cyanosis disappeared and respirations became slow and regular. The infant was placed in an incubator and oxygen was administered at 6 L. a minute. The tongue and tumor were kept moist with repeated applications of saline solution to prevent drying and cracking.

The following day, approximately 19 hours after birth, a Stamm type gastrostomy was done under 1 per cent procaine anesthesia for feeding purposes. The patient withstood the procedure without difficulty and continued to progress satisfactorily.

Thirty-six hours after birth respirations became labored intermittently. A roentgenogram of the chest showed a right pneumothorax with about 50 per cent collapse of the right lung, and some mediastinal emphysema. It was also noted that the tracheotomy tube was encroaching on the carina of the trachea and this was thought to be causing partial obstruction, which probably accounted for the mediastinal emphysema and pneumothorax. In addition, there was a tension pneumothorax on the left with mediastinal shift to the right. This was corrected by repeated aspiration of both hemithoraces, and no further difficulty was encountered.

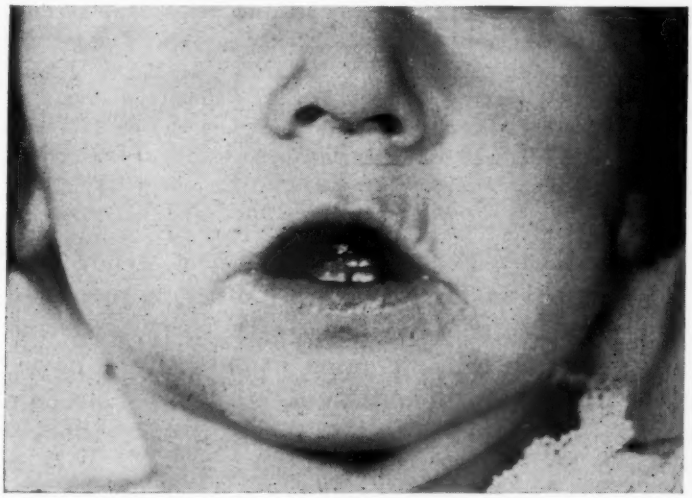

Fig. 4.—Front view of patient at age of 15 months. Slight peri-oral wrinkling still remains. Tongue is normal in size.

A roentgenogram of the tumor in situ (Fig. 2) showed widening and probable dislocation of the mandible by the mass. It contained several bony structures, including a long bone resembling the fibula, structures resembling the patella and femur or pelvic bones, and calcified structures resembling teeth.

For the next few days the tumor bled spontaneously, apparently from drying and cracking of the surface. A small blood transfusion of 10 c.c. per pound of body weight was given. Hematocrit was 36 per cent and hemoglobin 10.8 Gm. or 70 per cent.

Four days after birth, under rectal Pentothal anesthesia (0.2 c.c. of a 10 per cent solution per pound of body weight plus 25 mg. Pentothal intravenously) the tumor was excised (Fig. 3). It was not attached to the mandible, and was relatively easy to excise although it extended far down into the pharynx. Only the anterior one-third of the hypertrophied tongue was excised with the tumor. The tongue margins were sutured to the alveolar margins. At the end of the procedure the jaw and mouth could not be closed, and the tongue partially filled the open mouth.

Postoperatively the patient was given penicillin, 25,000 units, every 3 hours. The tracheotomy tube was removed 36 hours after excision of the mass, with no subsequent respiratory difficulty. Gastrostomy feedings were continued until the fourth postoperative

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day, when she was able to swallow liquids without difficulty. Thereafter, feedings of routine formulas were begun by mouth. Since the baby could not close her mouth sufficiently to appose the lips, sucking, of course, was impossible, and all feedings had to be given by a medicine dropper. Supplemental gastrostomy feedings were carried on for a few days after this. Sucking movements became stronger and stronger daily. The patient vomited the formula on several occasions and gain in weight was slow. However, the gastrostomy tube was removed 16 days after excision of the teratoma and the patient was discharged.

One month after discharge examination showed that the tongue was slowly decreasing in size, and the infant could now appose her lips and close her mouth. She had gained 12 ounces since birth. It appeared that she was going to be tongue-tied, but this could be easily remedied at a later date.

At the age of 6 months, the baby weighed 12 pounds, 14 ounces and was 62.5 cm. tall. The tongue was still decreasing in size and could be held behind the lips; that is, the tongue-tie noted previously appeared to be correcting itself with growth. The mother reported that the child had a great tendency to vomit, so thicker feedings were suggested. Other than this, the child appeared to be doing extremely well.

At the age of 14 months, the patient was gaining weight (weight 19 pounds, height 74 cm.), but the incisor teeth appeared carious. Roentgenograms of the wrists showed no evidence of rickets; results of the Mantoux test were negative. Development of the mouth, tongue, and jaw was nearly normal (Fig. 4). Roentgenograms of the mandible revealed a normal appearance. The child could protrude the tongue between the teeth and was speaking several words well with clear enunciation.

SUMMARY

A case of epignathus with survival of the host, believed to be the only case of its kind reported in the medical literature, is presented. Operative treatment included tracheotomy 5½ hours after birth, gastrostomy 19 hours after birth, and excision of the epignathus at the age of 4 days. At the time of this report the patient is 15 months old, has little or no deformity of the mouth, and is in good health.

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LYMPHANGIOSARCOMA IN POSTMASTECTOMY LYMPHEDEMA

A Case Report

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IN 1948 Stewart and Treves[1] described a new disease entity in which lymphangiosarcoma originates in the massive arms of women with postmastectomy lymphedema. They reported 6 cases. Later Ferraro[2] and McCarthy and Pack[3] each reported an additional case, bringing the total reported cases to 8. Since the nature of this tumor has eluded both surgeons and pathologists in the past, it must be assumed that other cases have gone unrecognized or misdiagnosed. As Stewart and Treves pointed out in their excellent description of this syndrome, the lesion may be confused from a clinical viewpoint with Kaposi's sarcoma, and from a small biopsy such a diagnosis is difficult to avoid. Because so few cases have been reported, and in order to stimulate further the recognition of this entity, the following case is reported.

CASE REPORT

L. B., a 53-year-old white woman, reported to the Hermann Hospital Outpatient Clinic on Jan. 2, 1938, with a 5 cm. mass in the lower outer quadrant of the left breast. The duration was indefinite. There were no palpable axillary lymph nodes. A diagnosis of carcinoma was made and the patient was given preoperative x-ray therapy. Irradiation totaling 10,427 r. units was administered to the left breast, left supraclavicular area, and left axilla. On March 25, 1938, a left radical mastectomy was performed. The pathologic report on the operative specimen was carcinoma of the breast with metastasis to one axillary lymph node.

Postoperatively the patient developed swelling of the left arm which gradually extended to the forearm and hand. She remained free of any evidence of recurrence of the carcinoma during the ensuing 12 years. Her lymphedema, however, became progressively worse, the left arm remaining enlarged to 2 to 3 times its normal size.

In April, 1950, the patient noted a small dark spot on the lateral surface of the left arm just above the elbow. The lesion grew larger and ulcerated. On Sept. 25, 1950, the patient reported to the Outpatient Clinic with a purplish-red tumor approximately 7 cm. in diameter just above the left elbow. Several small bluish-purple lesions varying in size from 5 mm. to 2 cm. surrounded the tumor. The surface of the tumor was ulcerated and crusted, and it bled easily. A small biopsy taken from the edge of the tumor was reported to be malignant melanoma. A chest roentgenogram was reported negative for metastasis. The patient was admitted to the hospital on Oct. 6, 1950, and a disarticulation amputation of the left upper extremity was performed.

The pathologist's report on the operative specimen was as follows:

Gross Specimen.—The specimen consisted of a left arm which had been disarticulated at the scapulohumoral joint. At 2 cm. above the elbow there was an elevated, dark, oval lesion on the skin measuring about 6 cm. in diameter and elevated about 1 cm. above the level of the skin. On section this lesion was seen to extend beneath the level of the skin,

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forming a total thickness of about 2.5 cm. The adjacent skin, including a cuff about 1 cm. around the lesion, was also indurated and showed pigmentation. Small black nodules and discolorations were seen scattered in the subcutaneous fat in the region of this tumor. One such nodule was in approximately the location of the epitrocheal lymph nodes. A section of this one was taken. No large axillary lymph nodes could be identified. The vessels showed no thrombosis nor any considerable thickening of their walls.

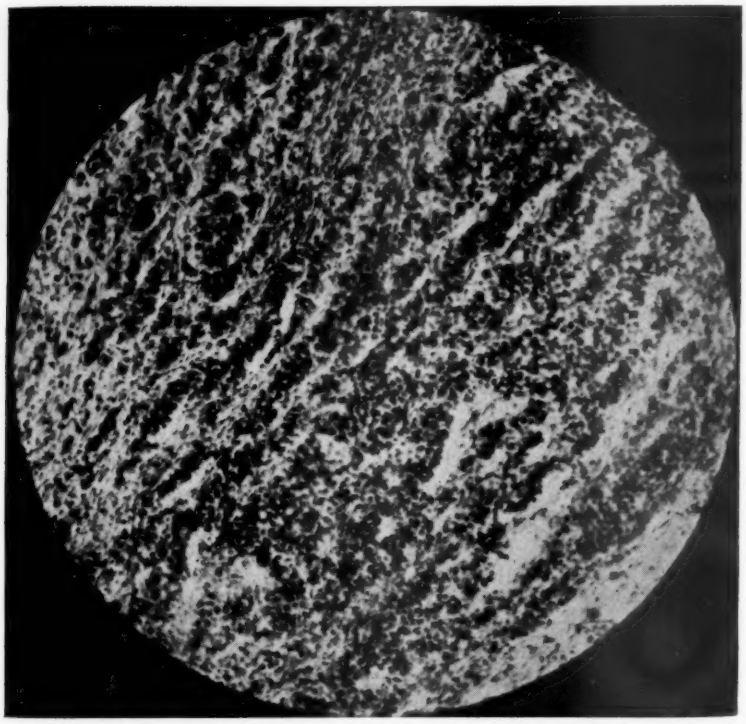

Fig. 1.—Adenocarcinoma of the breast. Original breast tumor composed of adenomatous cells arranged in cords and pseudogland formation in fibrous tissue.

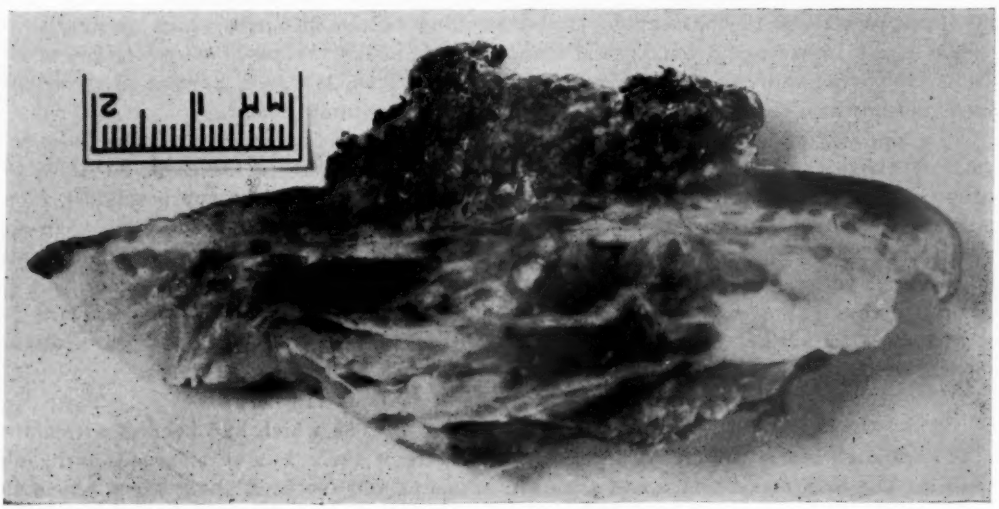

Fig. 2.—Lymphangiosarcoma. Gross cross-section of cutaneous lesion. Note deep pigmentation in subcutaneous tissue. Also note satellite lesions on each side of the main lesion.

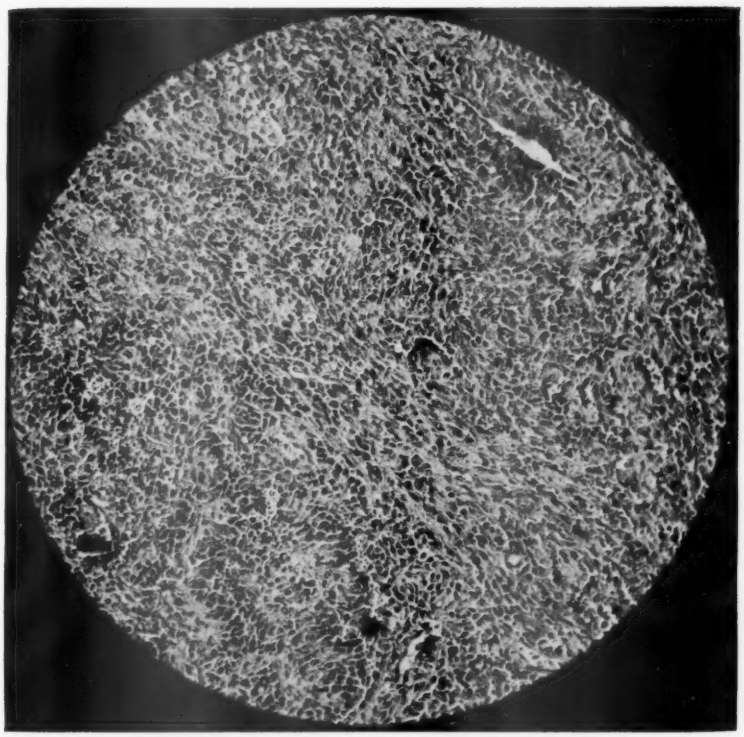

Fig. 3.—Lymphangiosarcoma. Low-power field of tumor proper. Note elongated angular cells giving whirled appearance of sarcoma. Several spaces are seen formed by tumor cells.

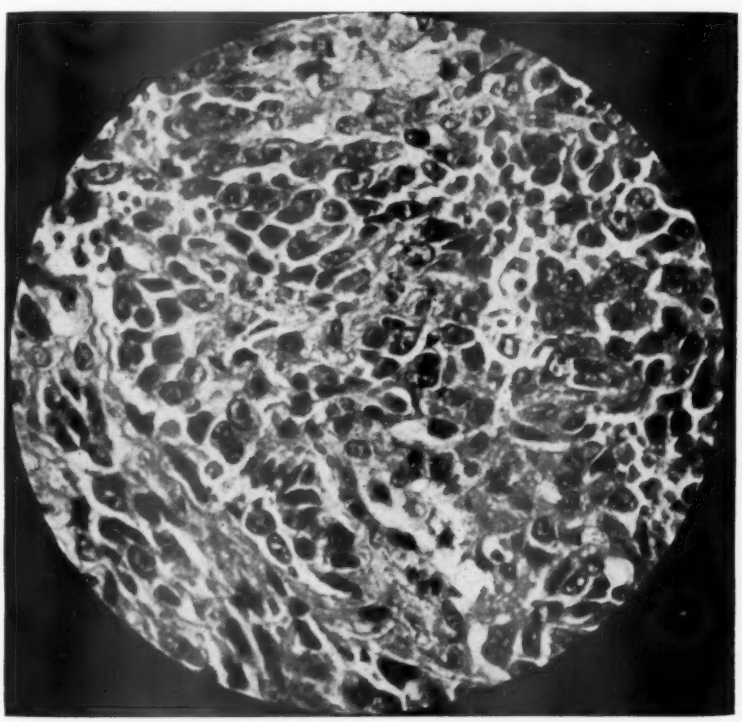

Fig. 4.—Lymphangiosarcoma. High-power field shows marked anaplasia of most cells. Some show definite endothelial pattern in forming lymph spaces.

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Microscopic Specimen.—There was a tumor composed of masses of large, elongated cells which formed spaces within the masses. Some of these spaces contained blood while others did not. There was much hemorrhage throughout the tumor proper; however, discrete metastatic nodules showed no blood within the spaces themselves. The nuclei of these cells were very large and vacuolated, but contained little chromatin. They were large and elongated. In some areas these were well formed and simulated endothelial cells; in others, they were totally undifferentiated. There was much pigment through the sections; however, this was all contained within phagocytes. Melanin and iron stains showed this pigment to be blood pigment.

Pathologic diagnosis was lymphangiosarcoma of the left arm.

The patient had an uneventful convalescence and was discharged from the hospital on her fourteenth postoperative day.

Fig. 5.—Recurrent lymphangiosarcoma. Photograph was taken March 20, 1951 (five months postoperatively). This recurrence is identical in appearance to the original lesion on the arm.

On October 30, a recurrent lesion, purplish red in color and 1 cm. in diameter, was discovered in the skin of the chest wall about 10 inches below the amputation stump. She refused to have biopsy performed, but was followed in the Outpatient Clinic.

During the next few months the nodules increased rapidly in size and number and when last seen on March 14, 1951, she had multiple lesions of various sizes studding the left chest wall, the largest of which measured 5 cm. in diameter and appeared identical to the original lesion on the arm.

Comment.—The clinical entity of postmastectomy lymphangiosarcoma was emphasized by Stewart and Treves and permits the clinician to make the diagnosis even before the pathologist's report is available. The sequence of radical mastectomy for carcinoma of the breast, immediate postoperative

lymphedema, and a long latent period (from 5 to 24 years in the 9 recorded cases, the average being 12 years) followed by characteristic lesions on the lymphedematous extremity is sufficient evidence to make the diagnosis.

As Stewart and Treves pointed out, the interesting feature of these cases is the relationship of the prolonged lymphedema to the etiology of the tumor. These authors discussed the question and postulated the presence of a continued systemic carcinogen as the probable mechanism rather than the prolonged lymphatic obstruction per se. There is no question of recurrence of the original breast carcinoma in any of the 9 reported cases, all having remained "cured" as regards the original lesion.

In the light of these reports, every patient undergoing radical mastectomy for carcinoma of the breast who develops lymphedema should be followed for a prolonged period of time because of the possible development of this sarcoma.

SUMMARY

1. A case of lymphangiosarcoma in postmastectomy lymphedema is reported.

2. The clinical entity by which the diagnosis can be made is emphasized.

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NEPHRECTOMY FOR HYPERTENSION

The Second Reported Ten-Year Cure

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BURKLAND, Goodwin, and Leadbetter[1] reported the first case of a patient maintaining a normal blood pressure for ten years following nephrectomy for hypertension. They stated that while many enthusiastic accounts of apparent cure had been recorded, few had stood the test of time, the hypertension returning a few months or so after surgery.

Burkland and associates operated on a 6-year-old Negro boy whose blood pressure previously had averaged 155/105, removing a hypoplastic ectopic pelvic kidney in which the main artery was partially occluded by a bundle of smooth muscle. The blood pressure had remained normal ever since.

It is indeed rare to read of the first ten-year cure of any disease, and I felt it a privilege to record the second reported case of a positive cure of hypertension following nephrectomy by presenting a healthy 44-year-old man in person before the Multnomah County Medical Society at its regular monthly meeting Dec. 6, 1950. The case was reported as a cure in Northwest Medicine[2] of March, 1944, at which time only seven of the seventy-seven attempts to cure hypertensive disease by removal of a unilaterally diseased kidney had been successful in reducing the elevated blood pressure.[3]

CASE REPORT

A. S., a 34-year-old logging railroad section foreman, was perfectly well until April, 1940, when there was increasing frequency of morning headaches. In November, 1940, there was a sudden onset of dimness of vision, and it seemed as if the field of vision fluctuated with each heartbeat. There was no history of lumbar pain, dysuria, hematuria, or pyuria, no edema, nocturia, or frequency. He was admitted to Holladay Park Hospital in Portland on May 21, 1941, as a candidate for sympathectomy after having been hospitalized in central Oregon for two months because of weakness and shortness of breath with daily severe headaches. He was rapidly losing vision.

Physical Examination.—The height was 5 feet, 8 inches, the usual weight about 170 pounds. The blood pressure was 224/124. He had plethoric facies. The abdomen was slightly obese. No tumors were felt. There was no lumbar pain or tenderness on percussion. Examination of the eye grounds* showed choked discs with edema, albuminuric streaks toward the fovea, blood vessels as behind a veil of edematous retina, and petechial hemorrhages.

Laboratory Findings.—Blood and spinal fluid Wassermann tests were negative; blood urea nitrogen was 12 mg. per cent; the urine was largely negative. On three trials there was ability to concentrate the specific gravity to 1020 to 1022. The phthalein per cent output was 45 and 15 with 310 c.c. and 115 c.c., respectively. Immediate spinal puncture was suggested to save the vision, 12 c.c. of fluid being removed under definite increased pressure; the blood pressure dropped from 206/130 to 174/120 and ranged from 170/108 to 180/120 for ten days.

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*By E. V. Ullman.

On May 26 cystoscopy disclosed that the left ureter could not be entered beyond 2 cm. The right kidney was catheterized, and an intravenous pyelogram showed the right kidney functioning promptly. The retrograde pyelogram showed the right pelvis and calices within normal limits; the left kidney pelvis was not visualized through the series.

Operation.—On June 3, under Nupercaine spinal anesthesia, a large, globular, dark cystic kidney was exposed which was aspirated of most of its 600 c.c. of reddish chocolate-colored fluid to facilitate removal of the organ; this was concluded uneventfully.

Daily record of the subsequent blood pressure to June 25 showed a range of 164/110 to 126/74. On June 18 the retinal edema in both eyes had disappeared, and there were no fresh hemorrhages.

Follow-Up Study.—In September the patient went to work in the woods as a carpenter's helper. On June 5, 1942, the tonsils were removed. On May 22, 1943, the blood pressure was 140/100. The man has worked as a welder, missing practically no working days up to the time of this present report. On Nov. 3, 1943, the eye grounds showed no evidence of sclerosis; there was no evidence of albuminuric streaks or spots, and the vision had returned to 20/20. The man reads without glasses, and the blood pressure on March 11, 1951, was 130/90.

Pathologic Report.—*

Gross examination: This examination revealed a cystic kidney, markedly dilated pelvis, nothing grossly that could be stated to be definitely kidney tissue.

Microscopic examination: Immediately under capsule of the kidney there were found small whorled structures that histologically were recognized as hyalinized glomeruli. Only a few nuclei remained in these. About these there were a few convoluted tubules containing polychromatophilic colloidlike material. These tubules were dilated and lined by a single layer of atrophic, flattened epithelium. The spaces between these tubules were formed of fibrous connective tissue, in which lymphocytes and plasma cells were to be found. The various tubular structures beyond this could not be followed in routine section, due to marked distortion of the pattern.

The large arteries of the muscular type near the hilus were thick walled, and in most instances the wall was of greater thickness than the diameter of the lumen. What were thought to be arcuate vessels showed a marked thickening and hyperplasia of the muscular layers so that the lumen was less than the thickness of the wall. In the cortical region, immediately adjacent to the hyalinized glomeruli, were small vessels, presumably arterioles. In these the ratio was reversed, and the wall thickness was about twice or more that of the diameter of the lumen. Also there was an endothelial proliferation so that in some instances the endothelial nuclei formed a pseudopalisade arrangement. Complete lumen obliteration from the endothelial proliferation was also found.

COMMENT

The glomeruli, the arterioles, and the arteries of this patient's hydronephrotic nonfunctioning kidney demonstrate as beautiful an example of arteriosclerotic ischemic kidney disease as one would expect to find in the typical case presenting albuminuric retinitis and hypertension. Here was a case with beginning disintegration and yet with the incongruity of normal blood urea nitrogen and of fairly normal kidney function to the usual observation. The cystoscopy was instituted as routine, preliminary to projected sympathectomy for hypertension and with no inkling of the startling findings of a dysfunctioning left kidney.

Certainly, these symptoms and findings were not due to renal insufficiency. Goldblatt's[4] experimental hypertension induced by renal ischemia and its prompt

*By Vinton D. Sneeden.

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cure by removal some time later of the induced ischemic kidney, proves, as does this clinical case, that the diseased kidney must be present in the body for hypertension to occur.

My case does not bear out Burkland and associates' assumption that emphasis should be placed on locating a lesion of the main renal artery as a cause of the elevation of the blood pressure, and the history and preoperative findings differ materially from the reported hypertension of infancy. At the same time, it is well to repeat here Sensenbach's[5] criteria for the selection of patients with hypertension for whom nephrectomy is indicated; (1) The diseased kidney should be functionless; (2) the opposite kidney should function normally; (3) the hypertension should be of short duration; and (4), other factors being equal, the younger (sic) the patient, the greater the chance of favorable results following nephrectomy.

CONCLUSION

The vascular changes found in the kidney of the patient were localized there and this dysfunctioning organ provided a hypertensive substance which was eliminated by operation. In every candidate for sympathectomy the status determination of each kidney cannot be by-passed.

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PYODERMA GANGRENOSUM

A Complication of Nonspecific Ulcerative Colitis

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(From the Surgical Service of The Mary Imogene Bassett Hospital)

ULCERATIVE skin lesions, usually multiple and at times spreading with alarming rapidity, form a serious though rare complication of nonspecific ulcerative colitis. In 1930, under the heading of pyoderma (ecthyma) gangrenosum, Brunsting and associates[1] reported 4 patients with ulcerative colitis who developed sloughing skin lesions. Since that time a number of other authors[2-5] have also reported the occurrence of this complication. The lesions have been described under a number of names, such as infectious multiple gangrene of the skin, impetigo gangrenosum, pyoderma gangrenosum, and ecthyma. They occur not only as a complication of ulcerative colitis, but in a wide variety of debilitating diseases, usually associated with chronic infection and malnutrition. Several reviews of the literature have appeared,[5-7] as well as more general articles mentioning the skin lesions associated with ulcerative colitis.[8-10] For the most part, occurrence of the lesions has been reported in dermatologic and medical journals, but since the complication may well arise in patients undergoing surgical treatment, it has been thought that the present report might be of interest.

Pyoderma gangrenosum, when found in association with chronic ulcerative colitis, usually occurs during a period of exacerbation of the disease. It appears first as a pustule or crop of pustules, most commonly on an extremity, but sometimes on the trunk. The initial pustules tend to break down and spread rapidly locally, while similar lesions are likely to appear soon on other areas of the body.

There is little agreement as to the etiological factors of pyoderma gangrenosum apart from the fact that the condition occurs in poorly nourished, debilitated individuals suffering from acute or chronic infections. A wide variety of organisms and combinations of organisms has been recovered from the ulcerative lesions, and it may be difficult in individual cases to assess the relative importance of the roles played by nutritional factors and pathogenic bacteria in the production and spread of the ulcers. McCarthy and Fields[2] recovered *Staphylococcus albus* and hemolytic streptococcus, which they considered acted in symbiosis; Brunsting and associates[1] also found both hemolytic streptococcus and staphylococcus organisms; *Pseudomonas pyocyanea* has been reported by Jankelson and McClure[5]; Montgomery[11] reported finding hemolytic streptococcus. Ricketts and co-workers,[7] on the other hand, found no pathognomonic flora; this was true in the case here presented. Meleney,[6] in his monograph on

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surgical infections, discussed the lesions as a subdivision of bacterial synergism; certainly bacteriologic studies of the skin lesions should include anaerobic cultures. Some authors (Felsen[12] and Cohen[3]) consider that the skin ulcers associated with ulcerative colitis are primarily a manifestation of avitaminosis.

CASE HISTORY

Mrs. E. P., a 30-year-old housewife, was admitted to the Bassett Hospital with a chief complaint of bloody diarrhea. The past and family histories were not remarkable. For the past 10 years she had suffered from chronic nonspecific ulcerative colitis, characterized by a number of acute exacerbations during which she had had abdominal cramps and diarrhea, with blood and pus in the stools. Five years ago, she had had a particularly severe attack, the weight falling to 75 pounds, and it was during this episode that she first noticed pain and swelling of the knee joints. The acute attack gradually passed, but recurred 2 years later in such severe form that she was hospitalized for one month. After a long convalescence, she improved considerably, and the weight increased from 72 to 130 pounds. The remission continued until 3 weeks prior to the present admission, when she began to suffer from migratory polyarthritis followed by progressively severe diarrhea. The stools increased in number to between 15 and 30 a day and were accompanied by abdominal cramps, considerable rectal pain, and increasing amounts of blood.

Physical examination showed a fairly well-nourished, pale young woman complaining of thirst. The abdomen was slightly distended; the liver and spleen were not felt. Rectal examination was impossible because of exquisite tenderness and stricture formation. The knee joints were swollen and warm. The blood pressure was 90/60, temperature 100° F., pulse 110, and respirations 24. The findings from a barium enema were those of long-standing ulcerative colitis with pseudopolyp formation.

Following admission to the hospital, the patient became progressively worse, in spite of energetic medical therapy. During the first 2 weeks she had a marked elevation of pulse and temperature. She received penicillin therapy, to which there was no response, and later streptomycin, which possibly caused some lowering of fever. With the deterioration of the patient's general condition, the arthritis became worse, and on the fifth hospital day skin lesions appeared in the region of the right knee and ankle and in the right cubital fossa where vein punctures had been carried out. It was at first thought that the lesions might have been a reaction to the tincture of iodine used in preparing the skin for vein puncture, but this idea was soon dispelled by the appearance of lesions in areas which had not been so treated. The lesions began as small pustules surrounded by an area of erythema; necrosis of the central portion soon followed, with a discharge of purulent material. They spread rapidly, and there was undermining of the skin edges. The appearance of the ulcers at various stages is shown in Figs. 1 to 4. Various local medications were tried in the first few days with little or no effect, among them gentian violet, penicillin topically, and Furacin. These preparations were eventually discarded in favor of bland petrolatum gauze dressings.

Laboratory data on admission were as follows: hemoglobin 6.8 Gm.; red blood cells 2,700,000; white blood cells 13,600, with polymorphonuclear leucocytes 68 per cent, lymphocytes 23 per cent, monocytes 8 per cent, and eosinophils 1 per cent; total serum proteins 5.1 Gm. per cent, albumin 1.9 Gm. per cent, globulin 3.2 Gm. per cent; blood urea 8.9 mg. per cent. The following laboratory data were obtained during the hospitalization. Warm stool examinations were carried out repeatedly and showed pus and blood, but no cysts or trophozoites. Stool cultures were negative for enteric disease organisms. Blood cultures taken near the height of the febrile episode showed no growth. The Wassermann was negative. A biopsy of the rectal mucosa on the tenth hospital day showed polypoid proliferation with marked inflammatory changes. The bacteriologic report on the first purulent material obtained from the ulcers was as follows: The smear showed rare gram-positive cocci; culture yielded *Staph. albus*, coagulase negative. Ten days later, no organisms were found in the smear; the only organisms grown from aerobic, anaerobic, and CO_2 incubation were the *Escherichia coli* group; incubation on Sabouraud's media was negative for fungi; biopsy showed an ulcer of the skin

in process of repair, with no evidence of specific inflammatory process. Studies of special preparations of the exudate from the ulcers did not show trophozoites or cysts. (It should be borne in mind that ulcers of the skin may occur also in amebiasis and *Amoeba histolytica* has been recovered from the skin lesions.[13-15])

Fig. 1

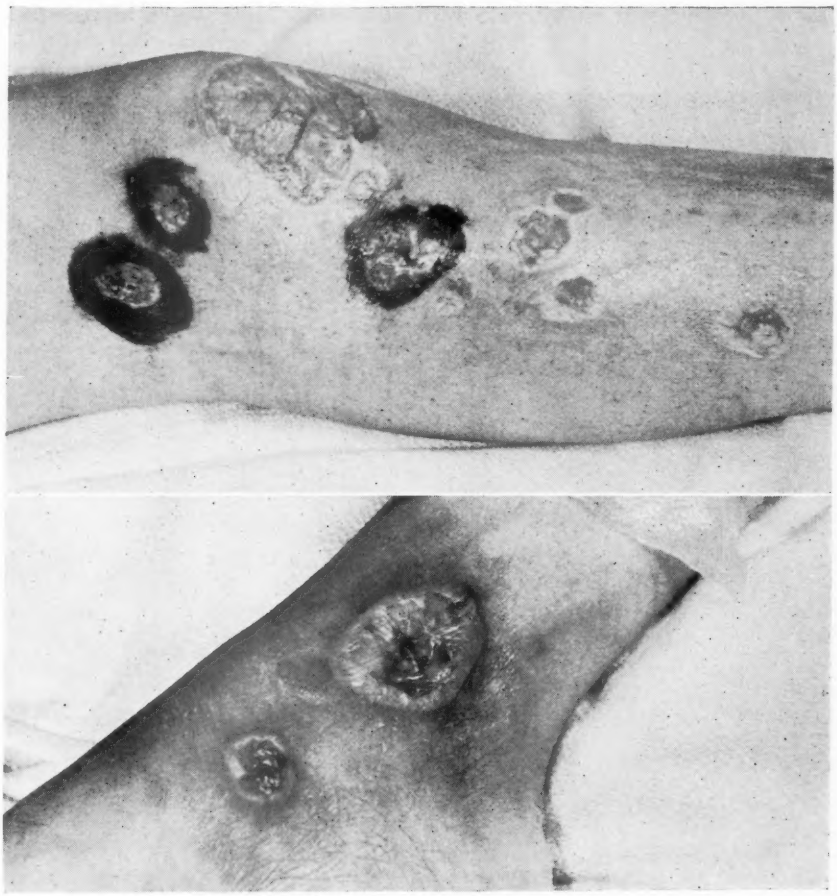

Fig. 2.

Fig. 1.—Skin ulcers (ringed with gentian violet) in the region of the right knee on the seventeenth hospital day, 5 days after the pustules were first noted.

Fig. 2.—Lesions of the ankle on the same day.

On the fourteenth hospital day an ileostomy was carried out. Following this, there was a gradual but steady improvement in the patient's general condition. The temperature returned to normal, and the diarrhea slowly subsided. Coincidental with the systemic improvement, the ulcerative lesions began to heal: Fig. 3 shows their appearance a few days before discharge from the hospital. On the sixtieth hospital day the patient was sent home for a period of recuperation prior to colectomy. The general condition was considerably improved, and the ulcers were healed. She was readmitted approximately 4 months later and underwent a subtotal colectomy. There was no recurrence of the ulcers during this admission. The patient was discharged on the twelfth postoperative day in good condition. A number of months later, she was seen in the outpatient department, when the ulcer scars were as shown in Fig. 4.

There is no general agreement as to the best management of pyoderma gangrenosum, aside from the belief that if the patient's primary disease and nutritional state are improved, the lesions tend to heal. Weiner[16] reported the successful treatment of a patient with sulfanilamide; on the other hand, Ricketts and colleagues[7] noted failure of their patients to respond to either sulfanilamide or penicillin. Numerous ointments and solutions have been advocated in the

Fig. 3. Fig. 4.

Fig. 3.—Ulcers in process of healing, on fifty-sixth hospital day.
Fig. 4.—Scars, one year later.

local treatment of the ulcers, and ultraviolet light has also been used.[7] In certain instances topical application and systemic administration of the appropriate antibiotics may be important, but the evidence in the literature is conflicting.

In the present case, after trying various topical applications, it was decided that bland dressings of petrolatum gauze were the most satisfactory. Whether or not the other local applications tried were of any value, it is difficult to say. It would seem that in this instance nutritional factors were more important in

the production of the ulcers than were the varying bacterial flora. The healing of the ulcers coincided with the general improvement in physical condition, and certainly therapy should include vigorous measures to improve the nutritional state of the patient. An adequate intake of food should be ensured, with special emphasis on proteins if the blood proteins are found to be low. Supplementary vitamins are usually indicated, and any existing anemia should be corrected.

SUMMARY

Ulcerative skin lesions (pyoderma gangrenosum) may complicate the course of nonspecific ulcerative colitis, as well as of other debilitating diseases.

There is general agreement that the poor nutritional status of the patient is a constant and important factor in the development of the skin ulcers. Some authors feel that this is the sole factor, while others give the leading role to a number of different organisms acting singly or in symbiosis. Treatment consists primarily of efforts to combat the ulcerative colitis and improve the nutritional status, while employing such topical and systemic medication as may be indicated by the bacterial flora of the ulcers.

In the case here reported it was felt that the patient's malnutrition and lowered resistance to infection rather than any specific pathogenic organisms were responsible for the development of the skin lesions.

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

THE EFFECT OF PARASYMPATHETIC OR SYMPATHETIC DENERVATION ON TOTAL STOMACH POUCH SECRETION IN DOGS

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THIS study of the changes in gastric secretion, caused by parasympathetic or sympathetic denervation, was preceded by observations of the influence of these types of denervation on experimental peptic ulcer formation. Vagotomy offered protection against histamine-provoked peptic ulcer in dogs,[29] while postganglionic sympathectomy, through excision of the celiac ganglia, led to an accelerated production of histamine ulcers.[28] Some explanation of these changes in ulcer sensitivity was found in the slowed gastric emptying caused by parasympathetic denervation[29] and in the increased adrenalin sensitivity caused by postganglionic sympathetic denervation.[27]

Several lines of evidence support the importance of the buffering and diluting action of food, almost constantly present in the stomachs of vagotomized dogs, as a protection against histamine-provoked ulcer. Lillehei, Lewis, and Wangensteen[29] have shown that the use of drainage operations to relieve the gastric stasis following vagotomy resulted in an increased incidence of histamine ulcer. They also noted that Urecholine, which induced a rapid emptying of the vagotomized stomach, led to a high incidence of histamine ulcers in vagotomized dogs. Further, it may be suggested that the relatively high incidence of histamine-provoked ulcer noted in dogs which were started on histamine shortly after vagotomy in contrast to the low incidence of ulcer in animals allowed a long postoperative recovery prior to a course of histamine injections was due in part to the absence of food in the stomachs of these dogs. These animals were usually not eating well when started on histamine, probably due to the general effects of the recent surgery. Finally, indirect evidence is found in the work of Fast, Friesen, and Wangensteen,[14] who observed that the Sippy regimen, in which the stomach contains food almost constantly due to frequent feeding, also protected against histamine-provoked ulcer.

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Celiac ganglionectomy causes a rather rapid gastric emptying, but this alone is not enough to account for the increased sensitivity to histamine ulcers which follows this operation. It has been suggested previously by Lewis and Wangensteen[27] that the increased ulcer susceptibility is due at least partially to an increased adrenaline sensitivity of the gastric vessels. Evidence to support this idea was found when they noted that the increased ulcer susceptibility produced by a celiac ganglionectomy was abolished by operations such as a thoracolumbar sympathectomy or adrenal medullectomy, which are known to result in a greatly reduced adrenaline production.

An essential further approach to the solution of this problem concerning the changes in ulcer sensitivity produced by parasympathetic or sympathetic denervation would be a study of the histamine-stimulated gastric secretion before and after each type of denervation. Such studies are described in this paper. In addition, to determine more completely the physiologic changes produced, basal secretion, 24 hour acid output, and action of cephalic and intestinal phase stimulants have been measured before and after parasympathetic and sympathetic denervation.

REVIEW OF THE LITERATURE

Effects of Vagotomy.—In the voluminous literature concerning the effects of vagotomy on gastric secretion, there is fairly general agreement that vagotomy is followed by a decrease in the basal secretory output and in the secretory response to food. These effects may not last indefinitely. Some disagreement exists as to whether or not the histamine secretory response is diminished after vagotomy.

In a brief, undocumented report in 1895, Fremont[16] described the first total stomach pouch preparation in dogs. Large amounts of acid-containing juice were secreted until the vagus nerves were cut, after which only a small amount of nondigestive juice was collected.

In Heidenhain pouch preparations, where the vagus innervation is lost, Pavlov[39] failed to observe a secretory response to sham feeding. He felt that there were important deviations from normal in the composition and secretion of gastric juice after vagotomy.

Litthauer,[33] in 1920, noted a lowering of acid output following vagotomy in Pavlov pouch dogs, and Thompsen,[45] in 1924, observed a fall in secretory output after vagotomy in dogs with gastrostomies. In 1925, Lim, Ivy, and McCarthy[32] found that mechanical distention was less effective in producing gastric stimulation in dogs when the vagus and sympathetic nerves were cut.

In 1929, Hartzell[21] studied the response of acid secretion to a test meal before and after vagotomy in dogs by using stomach tube aspiration. A reduction in acid production was found after vagotomy. However, Vanzant[48] in restudying 4 of Hartzell's dogs 2½ years after vagotomy noted a return nearly to normal of the acid secretory response to a test meal. The acid response to histamine was observed to be lower than in normal dogs; however, it was not studied before vagotomy in these same dogs.

Shapiro and Berg,[42] in 1932, also described a return to normal in the secretory response to a test meal after a period of depression lasting 6 to 12 weeks following vagotomy.

Ferguson,[15] in 1936, said that the acid tide following a test meal was not lowered after vagotomy in 6 monkeys.

Woodward and co-workers,[52] and Oberhelman and Dragstedt,[37] in 1948, described a reduction in the continuous secretory output and a decreased response to histamine in total pouch dogs after section of the vagus nerves. Anita and Ivy[2] also observed a reduced secretory response to histamine after vagotomy in dogs with gastric fistulas. However, in dogs with the vagus nerves intact and in other dogs with the vagus nerves divided, Hanson, Grossman, and Ivy[20] noted the need for similar minimal and maximal doses of histamine in order to cause minimal or maximal gastric secretory responses. They concluded that vagotomy does not alter the gastric secretory response to histamine.

The daily output of gastric juice in volumes of 1,000 to 2,600 c.c. which Dragstedt and Ellis[8] observed in total pouch dogs with the vagus nerves intact is in marked contrast to the 200 to 400 c.c. daily secretion in Ivy and associates'[24] total pouch dogs in which the vagus nerves had been cut. However, Webster and Armour,[50] in 1932, did not observe the high volume output described by Dragstedt when they studied dogs with gastric, esophageal, and duodenal fistulas in which the pylorus was divided and the vagus nerves were intact. These dogs secreted only 270 to 560 c.c. of juice a day, and the authors concluded that some abnormal mechanism caused the high daily output of juice in Dragstedt's dogs. Dragstedt, Woodward, Neal, Harper, and Storer[13] have recently (1950) clarified this problem by revealing that Dragstedt's original total pouch dogs had a portion of the antrum still attached to the duodenum, and then the antrum rather than the duodenum was anastomosed to the esophagus. When the antrum was left with the pouch and the duodenum anastomosed to the esophagus, total pouch dogs with the vagus nerves intact had a daily output averaging 550 to 632 c.c., more nearly the values found by Webster and Armour.

In human beings with peptic ulcer there has been general agreement that vagotomy lowers the continuous basal level of gastric secretion, but there has been some disagreement as to the relative power of humoral stimuli, such as histamine, before and after vagotomy. Dragstedt and associates,[9-11] Thornton, Storer, and Dragstedt,[46] Moore and co-workers,[36] Grimson and associates,[19] Stein and Meyer,[44] Kirsner, Levin, and Palmer,[26] Oberhelman and Dragstedt,[37] and Brooks and associates[4] all found a diminished gastric secretory output following successful vagotomy in human beings.

At first, Thornton, Storer, and Dragstedt[46] and Dragstedt,[11] in 1945, described no change in the gastric acid secretory response to histamine or caffeine after vagotomy in peptic ulcer patients. However, it was later reported from this same laboratory by Oberhelman and Dragstedt[37] that there was a decreased secretory response to histamine after vagotomy in both dogs and man. Moore and co-workers[36] agreed with Dragstedt's first opinion when they noted no change in the histamine response after vagotomy.

Though they expressed some doubt in the validity of their observations, Grimson and associates[19] described a diminished secretory response to both histamine and caffeine after vagotomy in human beings. Later, in a more extensive study, Stein and Meyer[44] observed the same diminished response.

Effects of Sympathectomy.—There is disagreement among the reported studies concerning the effects of sympathectomy on gastric secretion. Sympathectomy has been described as causing increased, unchanged, or slightly depressed gastric secretory output.

Gaultier,[18] in 1907, stated that section of the splanchnic nerves or excision of the celiac ganglia caused a hyperacidity. This was confirmed for section of the splanchnic nerves in dogs by Moll and Flint,[35] in 1928, and recently Schafer and Kittle[41] reported an increase in the level of gastric acid secretion following sympathectomy.

Other authors have found little or no change in secretory output following sympathectomy. Ishida,[22] in 1922, found no change in the secretion from a Pavlov pouch following sympathectomy. Thompsen,[45] in 1924, observed no change in the gastric secretion of dogs following splanchnicectomy and a rise in secretion in 2 of 3 dogs after celiac ganglionectomy. In cats, Brown[5] noted no demonstrable change in free or combined acid output after either the sympathetic chains or the celiac ganglia were resected, and Baxter[3] noted some depression in secretory output immediately after splanchnicectomy, with a later return to normal. Lim, Ivy, and McCarthy[32] also noticed a depressant effect; Heidenhain pouch preparations were found to respond to stimuli less effectively after the postganglionic sympathetic nerves were divided.

METHODS

Though Fremont[16] is generally credited with making the first total stomach pouches in dogs, Lim, Ivy, and McCarthy[32] in 1925, were actually the first to make extensive use of this type of pouch. In their dogs the vagus nerves to the pouch were divided. Dragstedt and Ellis,[8] in 1930, made total stomach pouches in which the vagus nerves were left intact, and it is this kind of a pouch that has been used in the present study.

The advantages of total pouch preparation over smaller pouch preparations, tube aspiration in the normal dog, or gastrostomies for the study of gastric secretion are readily apparent. With a total stomach pouch it is possible to collect all the secretion of the stomach without contamination by food, saliva, bile, pancreatic secretion, or intestinal juices.

In making a total pouch preparation the stomach was cut across at the esophagogastric junction and at the pylorus. Care was taken to leave no gastric mucosa attached to the esophagus and to leave no gastric antrum with the duodenum. Both vagus nerves were left intact to the stomach, and the sympathetic nerves, as they course along the blood vessels, were also left undisturbed. The cardiac end was inverted, and the pylorus was brought to the skin surface as a fistula. The alimentary tract continuity was re-established by anastomosing the esophagus to the first part of the duodenum end to end.

After a recovery period of 2 to 4 weeks, the various experiments to be described were performed; then the vagus or the sympathetic nerves were cut, and the tests were repeated on the same dogs. The experiments were done on each dog 3 to 7 times, both before and after nerve section. Because these animals were difficult to maintain and because they survived surgery poorly, a number of dogs were studied before nerve section which did not survive to be studied afterward.

Vagotomy was done through the bed of the eighth rib under pentobarbital sodium anesthesia. The esophagus was freed, and all vagal trunks were removed from the hilus of the lung to the diaphragm, after which the esophagus was carefully stripped of all pleura and superficial adventitia for a distance of 8 to 10 cm.

Celiac ganglionectomy was done by carefully stripping the celiac axis of all adjacent tissue from the aorta well out onto its 3 branches; both semilunar ganglia and their branches were removed in one segment. This was a postganglionic sympathectomy.

Preganglionic or thoracolumbar sympathectomy was done bilaterally in 2 stages. The eleventh and thirteenth ribs were resected, and the sympathetic trunk was removed transthoracically and retroperitoneally from the eighth thoracic through the second lumbar ganglia along with the splanchnic nerves as far as the celiac ganglia.

The dogs were fed a mixture of milk, cooked horse meat, and commercially prepared kibbled dog biscuit. If they did not eat large quantities of this mixture, a daily intravenous feeding of 500 to 1,000 c.c. of 5 per cent glucose in saline solution was given. To some dogs subcutaneous 5 per cent Amigen in 5 per cent glucose* was given, and salt tablets were fed to all the animals. Before testing, the dogs were fasted for 15 hours, and if daily intravenous feedings were being administered, they were omitted for the previous 24 hours.

On each specimen of gastric juice, volume was measured, and free and total acid were determined, using Töpfer's reagent (0.5 per cent alcoholic solution of dimethyl-amino-azobenzol) for free acid and phenolphthalein for total. Results expressed in degrees (clinical units, millinormality) were divided by 1,000 and multiplied by the volume to obtain the milliequivalents of acid per specimen.

EXPERIMENTS AND RESULTS

Effects of Vagotomy

1. Fasting Acid Output.—After the dogs had fasted 15 hours, specimens were collected at one-half hour intervals for 5½ hours; no stimulus was given. The results are shown in Fig. 1 in which averages of tests done on the same 4 dogs are compared before and after vagotomy. The data gained from 11 other dogs, studied only prior to nerve section, did not change the upper curves in Fig. 1. In the fasting unstimulated state there was a rather high continuous level of secretion by the vagus intact pouches which was almost

*Supplied by Mead Johnson & Company, Evansville, Ind.

completely abolished when the vagus nerves were cut. The difference between the means for milliequivalents of total acid in the fourth specimen before and after vagotomy appeared to be highly significant on statistical analysis, with a probability of .005.* The statistical probabilities for the various differences in acid output before and after vagotomy to be described are listed in Table I.

TABLE I. ACID OUTPUT OF TOTAL POUCHES BEFORE AND AFTER VAGOTOMY; SUMMARY OF STATISTICAL ANALYSES (SAME DOGS BEFORE AND AFTER)

GROUPS COMPARED	N*	$\bar{x}$	S.D.	P
Fasting before vagotomy	14	2.23	2.28	.0005
Fasting after vagotomy (meq. total acid in fourth specimen)	15	.02	.04	
Insulin tolerance before vagotomy	14	2.72	2.4	<.0005
Insulin tolerance after vagotomy (meq. free acid in sixth specimen)	13	.04	.46	
24 hour volume before	12	534	142	<.0005
24 hour volume after	13	103	39	
Histamine before	16	6.07	2.37	<.0005
Histamine after (meq. in first specimen after injection)	21	1.92	1.50	
Alcohol before	12	3.01	1.81	.0007
Alcohol after (meq. free acid in second specimen following alcohol)	15	1.01	.80	

*N = number of experiments
$\bar{x}$ = mean
S.D. = standard deviation
P = probability of difference in means being due to the errors of random sampling.

2. Insulin Tolerance Test.—Insulin hypoglycemia, which acts as a cephalic phase stimulant, was tested according to the method of Jemerin, Hollander, and Weinstein.[25] Three fasting specimens were collected at one-half hour intervals, and one blood sugar determination was made; one unit of insulin was then injected intravenously for each 3 to 5 pounds of body weight, and 5 more gastric juice specimens were collected at one-half hour intervals. Blood sugar determinations were made each 30 minutes for 1½ hours after insulin administration, and a fall in blood sugar below 50 mg. per cent was considered necessary for a satisfactory test. The results are summarized in Fig. 2; the stimulation of acid output brought about before vagotomy is shown to be completely abolished postoperatively. On statistical analysis the difference in mean free acid output before and after vagotomy, 1½ hours after insulin was given, is significant.

3. Twenty-Four Hour Acid Output.—Gastric juice was collected hourly for a 24 hour period, and the animals were fed once during this period at 12 noon, 2 hours after the test had been started. The meal was a mixture of 150 Gm. of cooked horse meat, 75 Gm. of kibbled dog biscuit, and 200 Gm. of milk. Water was allowed as desired, but no parenteral feeding was given during this period or for the 24 hours preceding it. As shown in Fig. 3, there was a consistently higher level of acid output before the vagus nerves were cut. This seemed to be as high during the night as during the day, and it was rather variable before vagus de-

*Formulas used in statistical analyses were taken from Treloar.[47]

nervation. The intestinal phase stimulation of the standard meal caused a small rise in acid output in the vagotomized pouches; however, the effect of the meal on the average acid output was not noted in the curve prior to vagotomy. An increase in acid output was occasionally seen in individual dogs following the meal, but the variability observed left the effect undetectable in the final curve of averages. At any rate, the increased acid output following the test meal in vagotomized dogs did not yield as much acid juice as was secreted continuously before vagotomy. The addition of 29 experiments on 11 others dogs tested only before vagotomy did not essentially change the character or the height of the before-vagotomy curve.

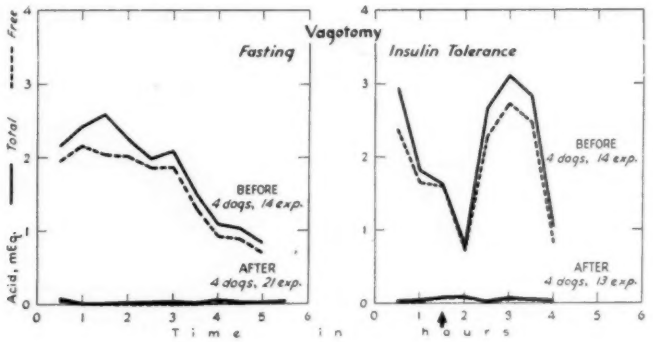


Fig. 1.

Fig. 2.

Fig. 1.—Fasting acid output of total pouch dogs before and after vagotomy. The acid was measured in milliequivalents per specimen: the same dogs were tested before and after vagotomy.

Fig. 2.—Acid output resulting from insulin hypoglycemia before and after vagotomy. The same dogs were tested before and after. The arrow indicates the time of stimulation in all figures.

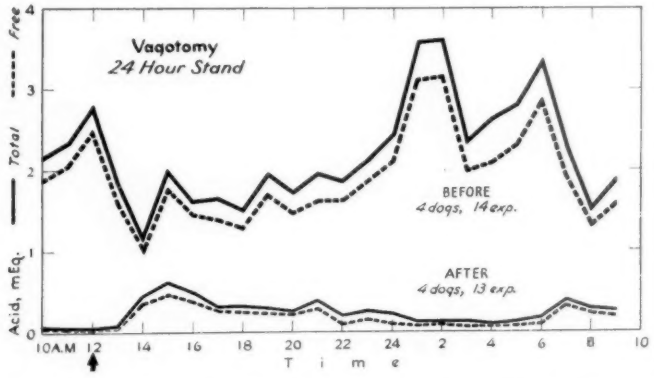


Fig. 3.—The 24 hour level of acid output in total pouch dogs before and after vagotomy. The same dogs were tested before and after vagotomy.

The average volume of pouch juice for 24 hours was 535 c.c., with a standard deviation of 142.5 in 12 experiments on 4 dogs before vagotomy, and 103 c.c., with a standard deviation of 39.0 in the 13 experiments on the same 4 dogs after vagotomy. The probability of this difference arising through the errors of random sampling is less than .0005 (Table I). The average 24 hour volume for the 43 experiments on all of the 15 dogs tested before denervation was 419 c.c. (S.D. = 178).

4. Histamine-Stimulated Secretory Output.—After 3 one-half hour fasting specimens were collected, 0.5 mg. of histamine base was injected subcutaneously, and collections were continued for 4 more hours. As shown in Figs. 4 and 5, histamine provoked a profound response which was much less after vagotomy. The difference in free acid secretion at the peak of the curves is statistically significant.

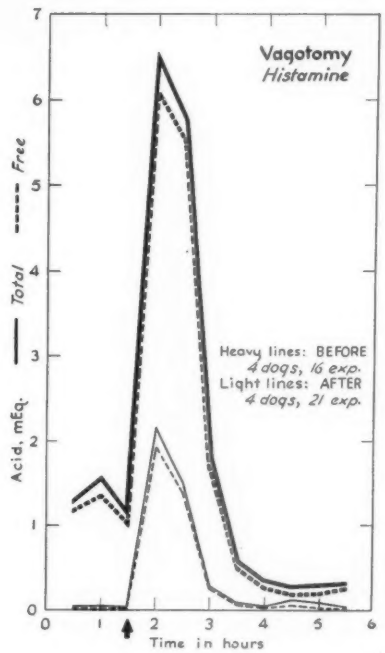


Fig. 4.

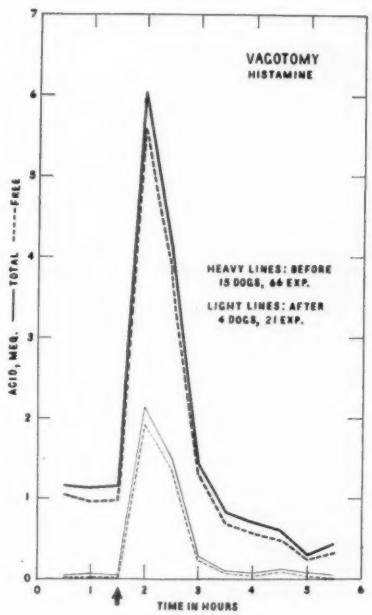


Fig. 5.

Fig. 4.—Histamine-stimulated acid output before and after vagotomy in total pouch dogs. The same dogs were tested before and after vagotomy.

Fig. 5.—Histamine-stimulated acid output before and after vagotomy in total pouch dogs. There were 15 dogs tested with nerves intact as compared with 4 after vagotomy.

It is important to note that the basic fasting level of secretion was much higher in the vagus intact pouches. As has been described by Lim[31] and by Lim, Matheson, and Schlapp,[30] the stimulated output depends directly upon the basal secretory output. It would, therefore, be expected that any procedure, such as vagotomy, which reduces the basal secretory output would result in an absolute reduction of the stimulated output to any secretory stimulus used. However, in comparison with its basal level, which is usually zero, in terms of free acid, for vagotomized pouches, the relative response expressed as output index (stimulated output divided by basal output) is greater in the vagotomized pouches.

When the histamine-stimulated response was graphed separately in terms of volume and of clinical units of free acid (the 2 measurements which were used to calculate the acid output in milliequivalents), the curves shown in Figs. 6 and 7 were obtained. The big response from histamine stimulation in vagus intact pouches was largely due to an increased volume of juice which had a high acid

concentration to begin with. In vagotomized pouches there was a greater relative increase in acid concentration to a high point which was still below that of the vagus intact pouches.

The effect of 30 mg. of histamine-in-beeswax (the dose used to provoke ulcer formation) on the 24 hour output of total stomach pouch secretion is shown in Table II. Two dogs were each tested once before vagotomy, and 2 different dogs each had one test after vagotomy. As might be expected, a greater volume of secretion was obtained from the total pouch dogs after histamine-in-beeswax than Code and Varco[6] obtained from Heidenhain pouch dogs.

TABLE II. TOTAL STOMACH POUCH OUTPUT FOR 24 HOURS AFTER INJECTION OF 30 MG. OF HISTAMINE-IN-BEESWAX INTRAMUSCULARLY

DOG	VOLUME (C.C.)	pH	FREE ACID (C.U.)	TOTAL ACID (C.U.)
Vagus intact	2,100	1.31	118	124
Vagus intact	1,850	1.29	128	136
Vagus sectioned	410			
Vagus sectioned	380	1.45	102	112

5. Alcohol.—Of the intestinal phase stimulants studied, only alcohol caused a consistent response; the others produced only slight, erratic increases in acid output. Alcohol was given as a 7 per cent solution. After 3 fasting specimens were collected, 50 c.c. were introduced into the duodenum with a tube, in an attempt to avoid cephalic phase stimulation, and collections were continued for 4 hours. The results are summarized in Fig. 8. As with histamine, the response was less after vagotomy. Statistical analysis of the difference between means of the free acid in milliequivalents secreted in the second specimen after stimulation showed a significant difference (Table I). Adding the data gained from 35 additional experiments on 10 other dogs, tested only before vagotomy, did not change the upper curve in Fig. 8 noticeably.

6. Meat Extract, Predigested Food, Raw Horse Meat, and Milk.—These substances, which might be expected to act as intestinal phase stimulants, were uniform in failing to produce a notable increase in average acid output; this was based on a number of tests either before or after vagotomy. In occasional individual experiments a slight rise in acid output was noticed.

For the meat extract stimulation 10 Gm. of Armour's beef extract were emulsified in enough water to make 50 c.c. and then injected into the duodenum through a tube.

Predigested food was obtained from the stomachs of vagotomized dogs sacrificed 10 hours after being fed a meal consisting of 200 Gm. of kibbled dog biscuit and 200 Gm. of raw horse meat; 20 Gm. of this partially digested mixture were made up to 50 c.c. by the addition of water and given to the animal with a tube.

Raw horse meat was fed to the dogs as a 200 Gm. meal over a period of 5 minutes or less.

For the milk stimulation, 50 c.c. were given by tube.

The results of the experiments with meat extract are summarized in Fig. 9. Curves for the other intestinal phase stimulants used were not reproduced here because they were all similar to those shown for meat extract. No distinct evidence of stimulation was shown, and all the curves were similar to the fasting curves of acid output described previously.

7. Intravenous Glucose in Saline Solution.—After 3 fasting specimens were collected, the animal to be tested was given 500 c.c. of 5 per cent glucose in normal saline solution intravenously within 5 minutes. The interesting results obtained are shown in Fig. 10. An increase in acid output occurred before vagotomy, but no response was found after vagotomy.

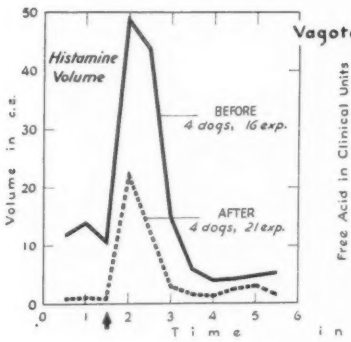


Fig. 6.

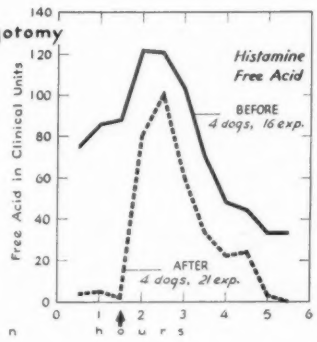


Fig. 7.

Fig. 6.—Histamine response in total pouch dogs before and after vagotomy in terms of volume of secretion.

Fig. 7.—Histamine response in total pouch dogs before and after vagotomy expressed as concentration of free acid in clinical units.

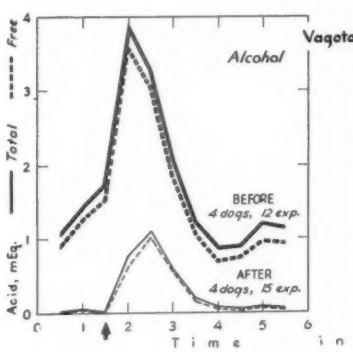


Fig. 8.

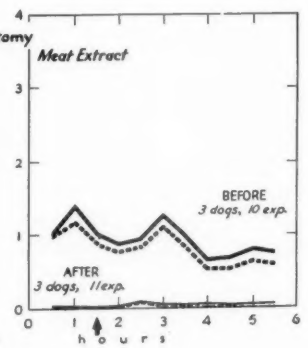


Fig. 9.

Fig. 8.—Acid secretory response to alcohol in total pouch dogs before and after vagotomy. The same dogs were tested before and after.

Fig. 9.—Acid secretory response to meat extract in total pouch dogs before and after vagotomy. The same dogs were tested before and after.

8. Tetraethylammonium Chloride (Etamon).—The effect of a ganglionic blocking agent, Etamon,* on acid secretory output was tested before and after vagotomy. After 3 fasting collections of juice, 2 c.c. of a 10 per cent solution were injected subcutaneously, and, as shown in Fig. 11, a depression of acid out-

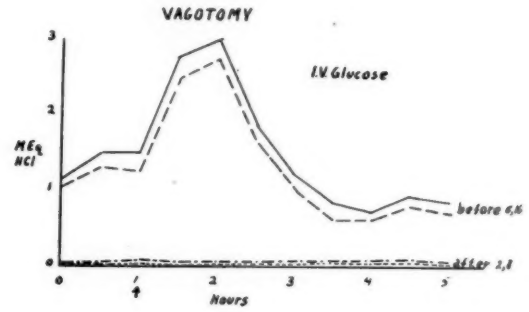
*Supplied by Parke, Davis & Company, Detroit, Mich.

put occurred in the vagus intact pouches. However, this "medical vagotomy" is transient, and even at the height of its effect the level of acid output does not fall as low as the constant basal level of acid output secreted by the vagus denervated pouches.

Postganglionic Sympathectomy (Celiac Ganglionectomy)

1. Fasting Acid Output.—After a 15 hour fast, specimens were collected at one-half hour intervals for 5½ hours. The average acid output in milliequivalents of free and total acid for 3 dogs tested before and after celiac ganglionectomy is graphed in Fig. 12. The curves overlap and show no effect of celiac ganglionectomy on the fasting basal output of acid.

Fig. 10.



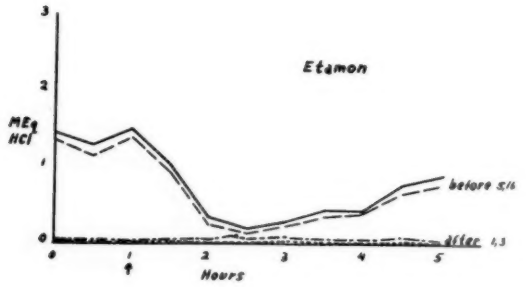


Fig. 11.

Fig. 10.—Acid secretory response to intravenous glucose in total pouch dogs before and after vagotomy.

Fig. 11.—Acid secretory response to subcutaneous Etamon in total pouch dogs before and after vagotomy.

2. Alcohol and Meat Extract.—Alcohol and meat extract, acting as intestinal phase stimulants, were given as described earlier. The alcohol response in terms of acid output remained unchanged by celiac ganglionectomy (Fig. 13). Though not graphed, no clear-cut effect of meat extract was demonstrated either before or after denervation.

3. Insulin Tolerance Test.—The effect of insulin hypoglycemia on gastric secretion was measured in one pouch dog before and after celiac ganglionectomy. The test was performed as described above. As would be expected, hypoglycemia produced a rise in acid secretion both before and after celiac ganglionectomy.

4. Twenty-Four Hour Output.—Hourly specimens were collected for a 24 hour period during which one test meal was fed as described above. Averages of the tests on 3 pouch preparations before and after celiac ganglionectomy are shown in Fig. 14. There is some overlapping of the curves and probably no significant change has been caused by celiac ganglionectomy, though the acid level seems to be slightly higher postoperatively. If all the data available on

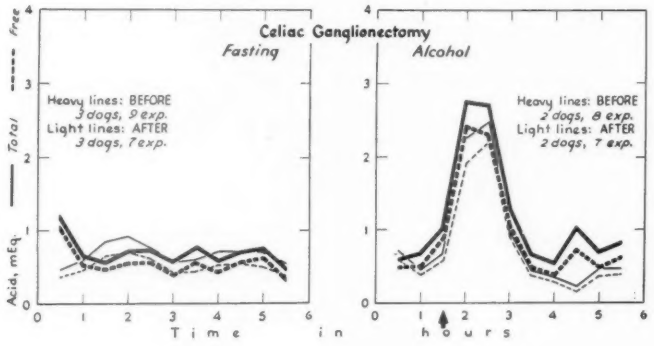


Fig. 12. Fig. 13.

Fig. 12.—Fasting acid output before and after celiac ganglionectomy in total pouch dogs. The same dogs were tested before and after denervation.

Fig. 13.—Alcohol-stimulated output before and after celiac ganglionectomy in total pouch dogs. The same dogs were tested before and after.

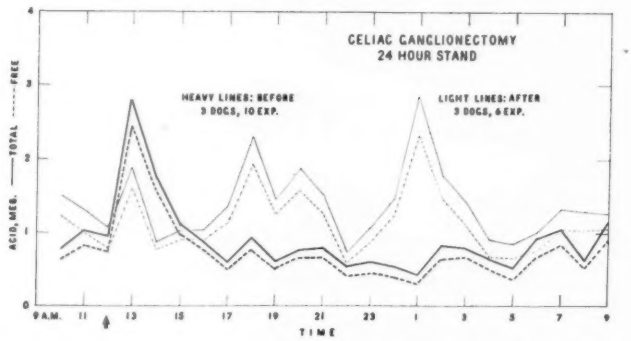


Fig. 14.—The 24 hour level of acid output in total pouch dogs before and after celiac ganglionectomy. The same dogs were tested before and after.

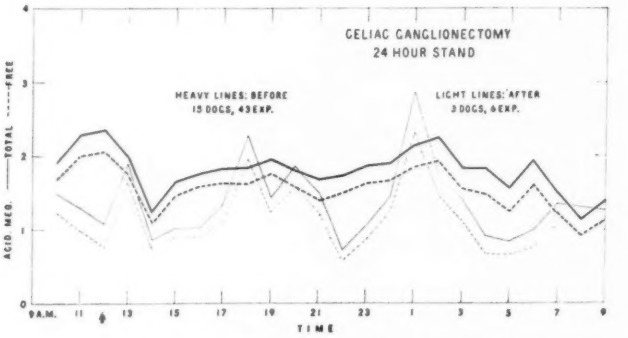


Fig. 15.—The 24 hour level of acid output in total pouch dogs before and after celiac ganglionectomy. There were 15 dogs with nerves intact as compared with 3 dogs after celiac ganglionectomy.

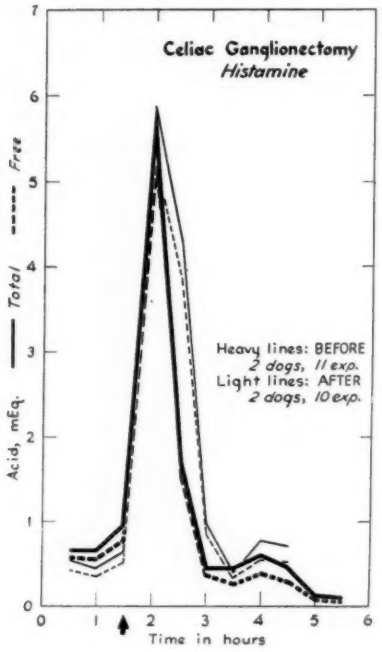


Fig. 16.

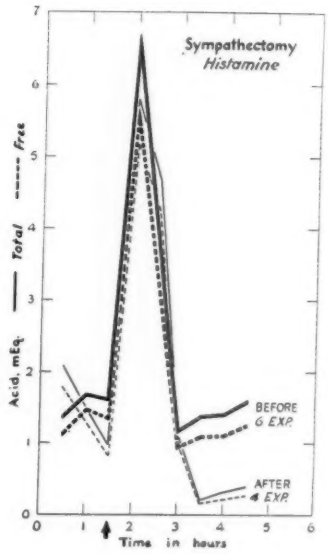


Fig. 17.

Fig. 16.—Histamine-stimulated acid output before and after celiac ganglionectomy. The same dogs were tested before and after.

Fig. 17.—Histamine-stimulated acid output of a total pouch dog before and after thoracolumbar sympathectomy.

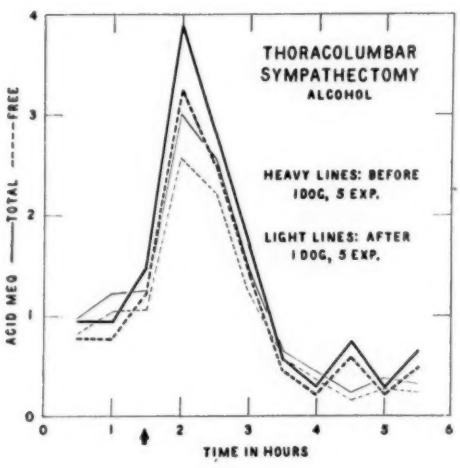


Fig. 18.

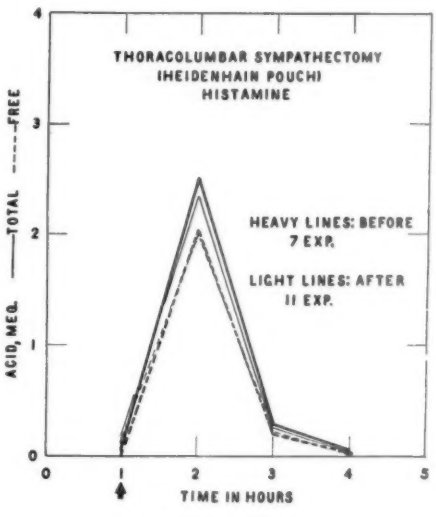


Fig. 19.

Fig. 18.—Alcohol-stimulated acid output of a total pouch dog before and after thoracolumbar sympathectomy.

Fig. 19.—Acid output response to histamine before and after thoracolumbar sympathectomy in one Heidenhain pouch dog.

pouches with nerves intact (43 experiments in 15 dogs) are compared with the acid level after celiac ganglionectomy in the 3 dogs studied, there is even less evidence of any change caused by celiac ganglionectomy (Fig. 15).

5. Histamine.—One-half milligram of histamine base in aqueous solution caused a striking increase in acid output both before and after celiac ganglionectomy, as shown in Fig. 16. There was no change effected by celiac ganglionectomy in the two animals studied.

Preganglionic Sympathectomy (Thoracolumbar Sympathectomy)

One total pouch dog was studied before and after bilateral thoracolumbar sympathectomy. Experiments as described previously were done to determine the fasting basal output and the response to histamine and alcohol stimulation. Figs. 17 and 18 are graphs of the results following histamine and alcohol stimulation which show no important changes in the character of the acid output curves following thoracolumbar sympathectomy. Similarly, the fasting basal output of acid was unchanged by thoracolumbar sympathectomy.

Some confirmation of these observations were found in experiments done with one Heidenhain pouch dog (vagus nerve branches to the pouch divided) which was tested before and after bilateral thoracolumbar sympathectomy. The basal fasting level was measured by collecting hourly specimens for 5 hours after a 15 hour fast. Histamine response was measured as acid output in hourly specimens collected for 3 hours after the subcutaneous injection of 0.5 mg. of histamine base. Five experiments before and 6 after thoracolumbar sympathectomy showed no change in the average fasting level of acid output. Histamine-stimulated acid secretion was also essentially unchanged by thoracolumbar sympathectomy (Fig. 19).

DISCUSSION

The secretory depression produced by vagotomy may be an important factor in the protection offered by this operation against histamine-provoked peptic ulcer. However, as has been described previously,[29] when the gastric stasis is relieved, ulcers may be produced in these animals with relative ease, even though the lowered secretory response to histamine presumably remains unchanged. This would suggest that gastric stasis is more important than secretory depression in causing the protection.

The results described here do not tell how long the secretory depression caused by vagotomy lasts. These total pouch dogs were studied no longer than 4 months after vagotomy. However, at that time, there was no suggestion that the effect of vagotomy on secretion was disappearing.

The abolition of the acid secretory response to insulin hypoglycemia after vagotomy in all of the pouch dogs is worthy of some comment. In dogs it is apparently relatively easy to effect this abolition by a careful vagus section; however, in man it appears to be much more difficult, and, frequently, a positive response to insulin will be found after the performance of what was

thought to be an adequate vagotomy.[1, 12, 49, 51] The possible occurrence of vagus fibers in the sympathetic trunks of man, as has been noted in cats,[40] has been suggested as an explanation of this observation.

The 24 hour volume of gastric secretion noted in the total pouch dogs with vagus nerves intact was similar to that found by Webster and Armour[50] in dogs with an esophagostomy, gastric fistula, duodenal fistula, and pyloric obstruction, and also similar to the values reported by Dragstedt and coworkers[13] in their most recent (1950) paper on total pouch dogs. After the vagus nerves were cut, our total pouch dogs secreted a 24 hour volume of gastric juice similar in amount to that noted by Ivy and associates[24] in total pouch dogs with the vagus nerves divided.

Dragstedt and associates,[13] in 1950, commented on the relatively large volume of gastric secretion in dogs with vagally innervated total pouches during fasting and said that these animals secreted almost as much juice in a 24 hour period when fasting as they did when a regular diet was eaten. We have also noted a high fasting output and have found no consistent increase in gastric secretion in these dogs after eating. The intestinal phase of gastric secretion seems to contribute little to the amount of gastric juice secreted by these dogs.

Intravenous glucose has usually been found to act as an inhibitor of gastric secretion.[7, 17, 38, 43] The rise in gastric acid after intravenous glucose administration, noted in our dogs with vagally innervated total pouches, may be due simply to the sudden infusion of a large amount of water. Ivy,[23] in 1918, found that intravenously administered water, in smaller amounts than we used, caused gastric secretion in man.

The abolition of a response to intravenous glucose after vagotomy might suggest that it acts as a cephalic phase stimulant and could be used as a test for the completeness of vagotomy. A brief trial of this method as a test following vagotomy in man gave unsatisfactory results.

The depression in gastric secretion which we observed in total pouch dogs following the injection of Etamon has also been observed by Lyons and coworkers[34] in man.

Neither pre- nor postganglionic sympathectomy caused any significant changes in the gastric acid output of total pouch dogs and one Heidenhain pouch dog. If small changes actually occurred following sympathetic denervation, the data presented have been insufficient to demonstrate them, and they must be much smaller in magnitude than the changes in acid output noted following parasympathetic denervation. The accelerated production of histamine ulcers, which has been described in dogs following celiac ganglionectomy, cannot be attributed to any change in the acid secretory response to histamine stimulation caused by this type of denervation.

CONCLUSIONS

1. With all the nerves intact to total stomach pouches in dogs, there was a relatively large amount of gastric juice with a high acid level secreted during

fasting and throughout a 24 hour period. There was a good response to cephalic, humoral, and one of the intestinal phase stimulants used.

2. After section of the vagus nerves to the total stomach pouches, there was a profound decrease in the output of acid during basal conditions, and the secretory response to the various stimuli tested was reduced.

3. Pre- or postganglionic sympathectomy did not change the acid secretory output of total stomach pouches during basal conditions or following various stimuli.

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Book Reviews

Surgical Forum, American College of Surgeons, Proceedings of the Forum Sessions, Thirty-Sixth Clinical Congress of the American College of Surgeons. Boston, Mass., October, 1950, Philadelphia, 1951, W. B. Saunders Company.

This volume is the first complete publication of the papers presented at the 1950 annual surgical forum held in conjunction with the Clinical Congress of the American College of Surgeons. The "Surgical Forum," originally conceived by Dr. Owen H. Wangensteen and developed under his guidance, was formalized by the regents of the American College of Surgeons in 1941. Since that time it has come to occupy a unique position in American surgery. This meeting has grown to be one of the highlights of the annual Clinical Congress of the American College of Surgeons and today provides a primary sounding board for the new and stimulating work of America's younger surgeons. In this volume, for the first time, all the papers of the 1950 meeting are made available. This publication and its progeny should achieve even wider acceptance and acclaim by the profession than the forum itself has received. For with this publication one can, in the quiet of his study, soberly digest and assimilate the wealth of new ideas and investigative facts that each year are presented by America's maturing surgeons. Moreover, the reader has the added advantage of being able to read a completed manuscript, whereas the essayist in presenting his material at the forum is limited in time (ten minutes) and may, of necessity, have had to hurry through important aspects of his paper to conform to the time limitation.

The volume includes all phases of surgical science and is divided into sections relating to (1) surgery of the lungs and esophagus, (2) surgery of the stomach, (3) surgery of the peritoneum, small and large bowel, and pancreas (4) liver and bile ducts, portacaval anastomosis, and kidney, (5) cardiac surgery, (6) blood vascular system and blood flow, (7) neurosurgery, (8) wounds and wound healing, tissue transplantation, antiseptics, and antibiotics, (9) water, electrolytes, protein, preoperative and postoperative care, fat metabolism, nutrition, and skin preparation, (10) blood transfusion, coagulation, shock, and hemorrhage, (11) malignancies and endocrines, and (12) anesthesia. Each paper is short, concise, and factual, with excellent tables, charts, and photographs to illustrate and clarify the data. Pertinent references follow each paper. This comprehensive syllabus of current surgical research will prove of real and lasting value to all students, teachers, and practitioners of surgical science.

The Hinge Graft or Ginglymus Implant. By Arnold K. Henry, M.B., Dubl., M.CH (Hon.), Cairo; F.R.C.S.I. Emeritus Professor of Clinical Surgery in the University of Egypt; Professor of Anatomy in the Royal College of Surgeons in Ireland. Pps. 64, with many illustrations. Hard. Price $3.00. Baltimore, Md. Williams & Wilkins Company, Publisher, 1950.

To those acquainted with Henry's "Extensile Exposure"[1] this little collection of cases and ideas needs neither review nor recommendation for his delightful style transcends the commonplace. A series of problems in reconstructive surgery form the background for the informal presentation of a variety of ideas and proposed solutions. The material is provocative rather than profound. It is especially directed toward surgeons engaged in repairing the ravages of trauma. The author is generous with his thoughts and, as he says, asks many "a burr-like question which I hope will stick and germinate in someone's cortex."

1. Henry, Arnold K. Extensile Exposure Applied to Limb Surgery, Edinburgh, 1945, E. & S. Livingstone.

Books Received

The receipt of books is acknowledged in this section and this treatment must be regarded as sufficient acknowledgment of the courtesy of the senders. Selections will be made for more extensive review dictated by the interests of our readers as space permits.

PEDICLE FLAPS IN PLASTIC SURGERY OF THE HEAD AND NECK. By Gordon B. New, M.D., F.A.C.S., and John B. Erich, M.D., F.A.C.S., University of Minnesota and Oral and Plastic Surgery, Mayo Clinic, Rochester, Minn. Soft. Price $3.00. Pp. 104, with 76 illustrations. Springfield, Ill., 1950, Charles C Thomas.

A.M.A.'s PRIMER ON FRACTURES. By the Special Exhibit Committee on Fractures in Cooperation With the Committee on Scientific Exhibit of the American Medical Association. Ed. 6. Hard. Price $2.00. Pp. 109, with 48 illustrations. New York, N. Y., 1951, Harper & Brothers.

STUDENT'S HANDBOOK OF SURGICAL OPERATIONS. By Sir Frederick Treves, Bart, G.C.V.O., C.B., LL.D., F.R.C.S. Revised by Sir Cecil Wakeley, K.B.E., C.B. Ed. 9. Hard. Price $5.50. Pp. 580, with 269 illustrations. Great Britain, New York, N. Y., Paul B. Hoeber, Inc.

LEHRBUCH DER RONTGENDIAGNOSTIK. By von H. R. Schinz, W. E. Baensch, E. Friedl, E. Uehlinger, Fünfte, vollig new bearbeitete und vermehrte Auflage. Soft. Price $16.50. Pp. 1406, with 2002 illustrations. Stuttgart, 1951, Grune & Stratton, Inc.

INTRODUCTION TO SURGERY. By Virginia K. Frantz, M.D., Associate Professor of Surgery, Columbia University, and Harold D. Harvey, M.D., Assistant Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University, and Associate Attending Surgeon, Presbyterian Hospital, New York. Ed. 2. Hard. Price $2.75. Pp. 233, with 12 illustrations. New York, N. Y., 1951, Oxford University Press.

TERMINAL CARE FOR CANCER PATIENTS. By Central Service for the Chronically Ill of the Institute of Medicine of Chicago, 343 South Dearborn Street, Chicago, Ill., 1951. Soft. Pp. 211, with 39 illustrations.

PHYSIOLOGY OF SHOCK. By Carl J. Wiggers, M.D., Sc.D., F.A.C.P., Professor of Physiology and Director, Department of Physiology, School of Medicine, Western Reserve University. Hard. Price $5.00. Pp. 459, with 56 illustrations. New York, N. Y., 1951, Commonwealth Fund.

ALLGEMEINE PATHOLOGIC. By von Prof. Dr. Med. Franz Büchner, Direktor des Ludwig Aschoff-Hauses, des Pathologischen Institus der Universitat Freiburg i. Br. Hard. Price D.M. 32.80. Pp. 528, with 424 illustrations. Germany, 1950, Urban & Schwarzenberg.